PUBLIC HEALTH REPORTS

VOL. 50 NOVEMBER 8, 1935 NO. 45

MORTALITY FROM CERTAIN CAUSES DURING THE FIRST HALF OF 1935 [1]

This report covers mortality in 31 States and Hawaii for the first half of 1935, with comparative data for the first half of the 3 preceding years. In addition to the death rate from all causes, rates are shown for 4 groups of diseases and 18 specific causes, some of which are included in the groups. Infant and maternal mortality rates per 1,000 live births are also shown.

The rates are computed from current and generally preliminary reports furnished by State departments of health. Because of some lack of uniformity in the method of classifying deaths according to cause, some delayed death certificates, and various other reasons, these preliminary rates cannot be expected to agree in all instances with final rates published by the Bureau of the Census. The final figures are based on a complete review and retabulation of the individual death certificates from each State. The preliminary rates given in the accompanying table are intended to serve as a current index of mortality until final figures are available.

In a group of 26 States [2] with an estimated population of about 90,000,000 the death rate for the first half year was the same in 1935 as in 1934, 11.5 per 1,000 (annual basis). The rates in 1933 and 1932 were less, 11.1 and 11.3, respectively. In approximately the same group of States the rate in the first half of 1931 was 11.8. In the first quarter the rate for 1935 was identical with that for 1934, and in the second quarter it was practically the same, 11.0 in 1935 and 11.1 in 1934. In both quarters the rates were less in 1933 and 1932 than in 1935 and 1934.

Infant mortality was 58 per 1,000 live births in the first half of 1935, as compared with 61, 59, and 59 in 1934, 1933, and 1932, respectively. Of 27 States with complete data for both years, in 5 the rate increased in 1935 over 1934, in 2 it remained the same, and in 20 it decreased.

[1] From the Office of Statistical Investigations, U. S. Public Health Service.

[2] See footnote to table for States included.

23190°—35——1

Tuberculosis mortality declined from 57.6 per 100,000 in the first half of 1934 to 56.3 in the first half of 1935. The decline in 1935 from the 1934 rate was less than in preceding years. Of 29 States with complete data, in 19 the rate decreased in 1935 and in 10 it increased. There was a very minor epidemic of influenza in the first half of 1935. The death rate from influenza, 30.1 per 100,000, is higher than in 1934 (22.0), which year was relatively free from this disease, but less than in 1933 (37.3) and 1932 (34.7), when minor epidemics occurred. In every State the influenza death rate in the first half of 1935 was higher than in the same half of 1934.

The pneumonia death rate in 1935 was the highest in the 4 years included, but was only slightly above that for 1934, when little influenza was present. In 9 States the rate was higher in 1935 than in 1934, and in 20 States it was lower. In both 1935 and 1934 the measles and whooping cough death rates were higher than usual, and the high pneumonia rates may be due in part to complications of these diseases in which the death certificate did not carry the preceding cause.

Measles and whooping cough death rates were both slightly less than in 1934. For measles, 14 of the 29 States had lower rates in 1935 than in 1934 and 15 had higher. For whooping cough, 20 of the 29 States had lower rates in 1935 than in 1934 and 9 had higher.

The typhoid fever death rate for the first half of 1935 was 1.0 per 100,000, as compared with 1.3, 1.4, and 1.8 for the first half of 1934, 1933, and 1932, respectively. Nineteen of the twenty-nine States had lower rates in 1935 than in 1934. The crude rate for diarrhea and enteritis under 2 years per 1,000 total population was also less than in preceding years. The declining birth rate, with the resulting smaller proportion of the population under 2 years of age, would produce a decline in this crude rate. Using as a population base the births in the current half year and the corresponding half of the preceding year, the rates per 1,000 live births in the group of States were 2.0, 2.4, and 2.2 for the first half years of 1935, 1934, and 1933, respectively.

The death rates from heart diseases, cerebral hemorrhage, and cancer increased in 1935 over 1934. The rate from nephritis was less than last year, and the rate from diabetes was practically the same. The majority of these degenerative diseases have for many years been either increasing or maintaining approximately the same level.

Mortality from certain causes in the first 6 months of 1935, with comparative data for the corresponding period in preceding years

[Estimated population July 1, 1935, 90,257,000]

State and period	All causes, rate per 1,000 population (annual basis)	Rate per 1,000 live births			Death rate per 100,000 population (annual basis)																			
		Total infant mortality	All except malformations and early infancy	Maternal mortality	Typhoid fever (1, 2)	Measles (7)	Scarlet fever (8)	Whooping cough (9)	Diphtheria (10)	Influenza (11)	Poliomyelitis (16)	Lethargic encephalitis (17)	Meningococcus meningitis (18)	Tuberculosis, all forms (23–32)	Cancer, all forms (45–53)	Diabetes (59)	Diseases of the nervous system (78–89)	Cerebral hemorrhage, apoplexy (82a, b)	Diseases of the circulatory system (90–103)	Diseases of the heart (90–95)	Diseases of the respiratory system (104–114)	Pneumonia, all forms (107–109)	Diseases of the digestive system (115–129)	Diarrhea and enteritis, under 2 years (119)	Nephritis (130–132)
26 STATES [1]																								
January to June—																								
1935	11.5	58	27	5.9	1.0	5.2	3.0	4.1	2.0	30.1	0.3	0.7	2.7	56.3	112.6	24.9	111.5	88.8	293.3	266.8	114.4	101.5	66.0	6.1	85.2
1934	11.5	61	28	6.1	1.3	6.2	2.7	5.0	2.0	20.0	.4	.6	1.0	57.6	110.2	25.0	108.8	84.7	294.9	263.5	112.4	98.8	67.3	7.2	89.4
1933	11.1	59	27	6.2	1.4	2.5	2.6	2.8	1.9	37.2	.3	.7	1.3	60.1	106.3	23.9	109.0	84.2	271.4	238.8	95.7	83.4	64.3	6.9	86.4
1932	11.3	59	27	6.3	1.8	2.6	2.9	4.8	3.5	34.7	.4	.7	1.7	65.2	104.0	23.8	111.0	85.5	268.2	234.5	106.5	93.9	65.7	7.1	89.7
January to March—																								
1935	12.0	63	31	6.1	.9	4.7	3.1	3.7	2.6	45.8	.3	.6	2.5	56.0	110.5	25.7	115.1	92.4	308.2	280.0	136.4	123.3	63.7	4.3	87.6
1934	12.0	64	30	6.0	.9	5.2	3.2	4.3	2.6	27.7	.3	.7	.9	57.2	108.1	26.5	113.7	88.6	312.2	278.4	133.9	119.3	63.9	5.7	92.9
1933	11.8	65	31	6.3	1.1	1.8	2.9	2.8	2.5	62.6	.3	.8	1.5	50.8	105.8	25.9	113.8	88.4	286.8	253.0	120.5	107.5	60.3	5.2	89.3
1932	11.9	60	28	6.4	1.9	2.0	3.2	4.9	4.6	43.5	.4	.8	2.0	64.9	103.6	25.5	114.6	89.1	281.4	246.3	130.0	115.8	63.2	5.5	93.2
April to June—																								
1935	11.0	54	23	5.7	1.2	5.8	2.8	4.5	1.5	14.6	.3	.8	2.9	56.6	114.6	23.1	108.0	85.3	278.5	253.8	92.5	80.0	68.2	7.9	82.8
1934	11.1	59	25	6.1	1.8	7.2	2.3	5.7	1.5	12.4	.5	.5	1.0	58.0	112.4	23.6	103.9	80.9	277.8	248.8	91.1	78.5	70.6	8.7	86.0
1933	10.5	54	22	6.0	1.7	3.1	2.4	2.8	1.4	12.4	.4	.7	1.2	60.4	106.9	21.9	104.3	80.0	256.2	224.8	71.1	59.6	68.2	8.5	83.6
1932	10.8	58	26	6.3	1.7	3.1	2.7	4.8	2.4	26.0	.4	.6	1.4	65.4	104.3	22.2	107.3	82.0	254.9	222.7	83.0	72.0	68.3	8.8	86.1
JANUARY TO JUNE																								
Alabama:																								
1935	10.7	(2)	(2)	(2)	1.9	9.9	.5	6.4	2.8	72.6	.2	.6	1.2	65.5	61.3	9.2	92.2	68.7	148.5	136.3	116.0	107.5	61.5	15.1	84.8
1934	10.7	76	48	6.0	2.2	18.1	.4	15.4	3.8	38.4	.4	.6	.4	67.6	57.4	11.3	90.9	65.1	159.2	147.3	110.6	102.0	71.6	17.9	79.2
1933	9.5	67	42	6.7	2.8	1.2	.4	6.7	2.5	47.7	.4	1.3	.4	71.1	55.1	9.2	83.9	57.1	135.4	124.4	69.9	64.1	58.4	14.1	79.0
1932	10.0	62	39	7.8	4.0	.4	1.1	7.2	4.1	40.7	.3	.7	.6	79.3	54.1	8.0	88.7	59.3	122.1	112.7	88.9	82.7	61.1	11.9	81.4

[1] States included are Alabama, California, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Louisiana, Maryland, Michigan, Minnesota, Montana, Nebraska, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Tennessee, Virginia, West Virginia, Wisconsin. Includes all of the States with available data for the 4 years covered by this summary. For a few causes, 1 to 3 States were omitted because of missing data.

[2] Data not available.

Mortality from certain causes in the first 6 months of 1935, with comparative data for the corresponding period in preceding years—Continued

State and period	All causes, rate per 1,000 population (annual basis)	Rate per 1,000 live births		Death rate per 100,000 population (annual basis)																				
		Total infant mortality	All except malformations and early infancy	Maternal mortality	Typhoid fever (1, 2)	Measles (7)	Scarlet fever (8)	Whooping cough (9)	Diphtheria (10)	Influenza (11)	Poliomyelitis (16)	Lethargic encephalitis (17)	Meningococcus meningitis (18)	Tuberculosis, all forms (23–32)	Cancer, all forms (45–53)	Diabetes (29)	Diseases of the nervous system (78–89)	Cerebral hemorrhage, apoplexy (824, b)	Diseases of the circulatory system (90–103)	Diseases of the heart (90–93)	Diseases of the respiratory system (104–114)	Pneumonia, all forms (107–109)	Diseases of the digestive system (115–129)	Diarrhea and enteritis, under 2 years (119)	Nephritis (130–132)
JANUARY TO JUNE—con.																								
California:																								
1935	11.8	51	21	4.7	.9	1.6	1.2	.9	1.7	11.7	.4	.4	2.1	76.3	131.4	25.2	108.0	82.1	362.4	319.8	111.2	82.8	67.5	72.0	5.2	82.4
1934	11.2	54	24	4.7	1.1	2.3	1.5	3.9	1.7	7.1	1.5	.4	.9	79.2	128.2	22.1	103.3	77.3	324.9	290.4	100.1	176.9	64.3	160.0	8.2	70.9
1933	11.7	55	24	5.1	.9	2.5	1.4	3.3	2.1	21.9	.1	.5	1.6	81.3	127.4	24.0	117.8	87.0	320.7	286.2	89.3	107.3	76.4	91.8	4.8	83.7
1932	11.1	54	24	6.2	1.1	1.6	1.0	2.3	3.4	13.0	.8	.5	1.7	87.1	119.5	21.3	107.6	79.8	294.8	260.5	82.2	(†)	69.1	102.4	6.7	86.2
Connecticut:																								
1935	10.9	47	(*)	5.6	.7	3.4	2.0	1.3	.9	12.3	.2	.5	1.1	47.2	123.8	34.6	(†)	(†)	483.7	427.6	224.7	(†)	90.0	(†)	1.5	92.7
1934	10.9	55	(*)	5.6	.5	.2	1.0	1.2	.4	10.1	(*)	.4	.7	46.8	122.0	28.6	(†)	(†)	487.1	428.4	185.1	163.2	88.7	96.4	3.9	94.9
1933	11.1	52	(*)	7.5	.5	1.0	2.0	2.3	1.2	35.7	(*)	.9	.5	54.0	130.6	25.3	(†)	(†)	428.6	353.6	140.9	131.6	93.1	107.1	4.5	92.0
1932	10.8	55	(*)	6.1	.7	1.8	1.7	3.9	1.0	22.5	.4	1.0	1.0	54.4	114.1	29.1	(†)	(†)	417.5	402.2	344.1	184.5	162.3	86.8	3.9	94.6
District of Columbia:																								
1935	18.2	62	29	4.9	1.6	(†)	3.2	.4	4.5	21.4	(*)	1.6	23.8	135.4	183.6	34.0	153.2	120.4	483.7	427.6	230.1	96.5	81.4	94.1	16.2	111.1
1934	17.5	68	38	3.1	.4	19.4	2.0	11.7	1.2	10.9	.4	1.2	.8	129.6	148.2	37.7	150.2	113.4	487.1	428.4	236.3	185.1	163.2	96.4	14.3	124.9
1933	16.5	64	26	5.0	1.2	1.2	4.9	.4	1.6	15.5	(*)	.4	2.9	135.3	153.2	27.7	167.0	127.1	428.6	353.6	191.4	140.9	131.6	107.1	9.4	122.2
1932	17.0	69	31	8.5	.8	(*)	4.4	5.7	4.8	17.8	.4	2.0	4.8	128.4	156.3	29.9	165.6	117.5	402.2	344.1	188.9	184.5	162.3	86.8	4.8	158.7
Florida:																								
1935	12.7	69	40	9.1	2.3	3.4	(*)	3.9	3.9	59.2	.6	(*)	.6	57.2	86.0	22.5	124.2	98.4	250.9	230.1	96.5	81.4	94.1	16.2	111.1
1934	12.9	70	37	8.0	3.1	9.6	.4	5.6	3.6	30.5	.5	.5	.1	62.1	81.6	17.1	130.8	99.0	255.8	236.3	104.5	90.6	94.7	14.3	104.5	124.9
1933	12.0	70	35	11.4	4.6	.1	.1	2.9	3.8	61.5	.5	.8	.8	90.6	82.1	15.9	121.3	92.6	208.8	191.4	76.9	65.2	73.6	8.2	122.2	122.2
1932	11.5	65	34	9.8	6.2	1.0	.6	1.8	5.2	35.8	.6	(*)	(*)	73.6	76.3	17.0	122.0	104.7	204.2	188.9	73.1	62.6	92.3	14.3	108.4	108.4
Georgia:																								
1935	11.3	75	(*)	8.0	3.7	1.5	.7	7.0	3.9	60.7	.7	.1	1.0	58.4	53.0	12.4	108.7	75.2	174.4	163.4	128.9	118.8	77.4	16.5	107.1	107.1
1934	11.9	88	(*)	8.2	5.6	32.5	.5	12.0	4.2	44.2	.6	.3	.7	60.0	53.9	12.9	113.4	74.4	180.7	165.9	139.5	128.5	74.2	17.2	104.5	104.5
1933	10.0	72	(*)	8.0	4.4	1.7	.4	6.4	2.7	66.2	.9	.1	.4	57.8	51.3	11.6	101.1	71.0	130.7	121.3	94.2	86.1	61.5	12.5	106.0	106.0
1932	10.7	70	(*)	9.7	8.6	.5	1.2	4.0	4.0	43.4	1.2	.6	1.0	67.7	48.8	11.9	118.4	81.6	143.7	132.2	110.4	101.1	62.6	10.6	111.9	111.9
Hawaii:																								
1935	8.1	68	42	5.4	2.4	(*)	(*)		3.3	7.6	(*)	(*)	1.4	75.3	63.4	15.2	61.1	46.4	119.8	111.2	82.8	72.0	68.6	24.1	70.5	
1934	9.4	60	60	6.6	3.9	.5	(*)	16.5	1.0	20.9	.5	(*)	1.5	83.1	62.2	18.0	56.4	36.9	112.2	100.1	176.9	160.0	83.0	35.0	60.3	
1933	9.5	79	50	5.3	8.0	.5	(*)	16.5	2.0	9.5	1.0	(*)	1.0	104.8	70.9	14.0	68.4	48.9	118.3	107.3	107.3	91.8	102.3	44.9	77.9	
1932	9.9	92	(*)	9.7	2.0	12.8	.6	2.0	7.2	5.1	1.0	.5	4.6	96.7	67.0	8.7	(†)	50.1	(†)	98.2	(†)	115.6	116.2	57.8	64.5	

Idaho:																									
1935	10.9	61	25	7.3	1.4	2.7	5.0	10.8	.5	25.2	.5	3.2	5.4	27.5	71.6	14.4	98.1	72.9	225.5	173.8	141.8	123.8	64.8	.9	31.1
1934	10.8	50	32	5.5	2.7	6.3	3.6	3.1	3.1	19.3	1.8	1.3	3.1	35.9	73.7	11.2	113.6	82.6	209.7	178.3	142.4	127.6	64.2	2.7	40.9
1933	10.0	52	17	1.9	5.0	.9	(1)	.5	2.3	28.4	(1)	(1)	1.4	30.2	87.3	11.3	113.4	77.4	209.3	175.6	95.4	78.8	59.0	.9	36.0
1932	9.6	66	38	5.6	2.2	(1)	.9	(1)	3.6	21.5	.4	1.3	3.1	33.7	62.8	12.6	119.4	90.2	205.6	166.1	98.8	87.1	57.0	2.2	47.1
Illinois:																									
1935	11.3	54	22	5.5	.7	7.2	7.7	3.3	2.8	24.6	.2	.6	3.7	54.0	124.8	25.1	98.5	74.2	306.0	284.4	107.7	96.8	68.6	3.5	100.2
1934	11.4	54	21	5.3	.8	4.3	4.9	3.4	1.5	13.1	.2	.5	2.1	54.2	123.0	29.7	102.2	73.5	304.5	282.1	101.0	90.0	70.1	4.6	109.2
1933	11.0	58	24	5.5	.8	1.2	4.9	.7	1.7	25.0	.4	.6	4.4	56.8	116.9	27.9	106.9	80.6	280.3	261.4	89.0	76.4	66.9	5.1	106.6
1932	10.9	58	25	5.5	.9	1.0	4.7	4.5	3.2	29.8	.5	.5	2.4	58.6	112.5	27.2	102.2	75.5	257.0	238.0	94.0	80.0	69.9	4.9	115.2
Indiana:																									
1935	11.9	59	26	5.8	.5	5.0	4.3	5.7	2.7	38.3	.1	.6	3.8	51.6	114.1	15.2	(1)	133.5	(1)	278.6	(1)	111.6	(1)	3.2	71.6
1934	12.8	57	25	6.3	1.8	11.5	4.5	5.5	2.7	26.9	.3	.4	.9	55.4	111.2	19.2	(1)	126.4	(1)	274.4	(1)	97.2	(1)	3.9	81.7
1933	11.6	57	25	5.4	1.8	.6	2.6	2.2	3.3	48.1	.4	1.3	2.0	57.7	106.7	16.3	(1)	112.9	(1)	187.3	(1)	80.5	(1)	5.8	79.6
1932	11.8	59	26	5.6	1.0	.8	3.1	7.8	4.3	49.9	.2	.9	5.8	63.5	106.1	16.9	(1)	115.0	(1)	180.6	(1)	103.6	(1)	6.9	71.8
Iowa:																									
1935	10.9	55	25	5.4	.8	12.1	3.1	1.6	2.0	33.1	.4	.8	2.1	28.2	128.5	23.6	134.0	108.9	269.7	242.7	109.9	99.9	57.0	3.1	65.3
1934	11.0	56	22	6.2	.5	3.9	2.8	4.4	1.9	26.9	.1	.9	1.3	25.6	122.9	24.9	153.3	120.4	249.7	217.0	112.3	98.2	59.6	3.0	67.8
1933	10.8	55	24	4.9	1.0	.3	1.5	2.3	1.2	56.4	2.3	1.1	2.0	26.6	123.4	21.3	143.8	116.8	256.0	201.4	113.0	91.5	52.0	1.5	45.2
1932	10.6	51	21	3.5	.8	.3	2.0	1.9	1.6	39.8	1.1	1.1	1.1	29.1	112.0	16.5	145.4	115.1	260.7	213.2	117.2	93.8	57.3	2.0	49.1
Kansas:																									
1935	11.3	56	28	5.5	.4	22.4	2.5	3.5	2.0	49.7	.2	1.5	2.9	29.3	108.4	21.7	119.8	97.0	258.8	231.3	124.9	113.5	67.3	4.6	90.9
1934	10.3	50	20	6.3	.6	3.2	2.6	6.8	2.0	25.9	.1	1.4	1.5	28.4	105.1	25.3	117.2	93.2	236.7	209.8	82.9	73.4	70.6	4.0	97.4
1933	11.3	62	30	5.8	1.0	1.4	1.9	3.1	2.3	80.2	.2	.4	1.5	33.9	104.6	25.8	134.9	111.7	222.9	202.5	88.1	76.5	66.7	4.9	100.8
1932	10.2	49	19	6.7	.7	2.4	1.8	2.4	3.5	46.0	.2	.6	2.0	34.6	104.6	22.4	124.7	102.9	203.1	182.3	68.4	59.8	62.0	5.0	104.5
Louisiana:																									
1935	10.9	77	42	7.7	7.2	9.9	.6	3.1	4.5	33.2	.6	.6	.9	76.6	73.3	14.7	90.6	67.2	200.2	176.2	106.7	95.7	79.9	15.8	105.7
1934	10.5	78	49	9.8	8.1	11.2	1.1	12.0	4.7	27.7	.3	.2	.8	71.5	72.8	13.2	83.0	56.3	213.5	193.2	94.5	81.6	70.2	20.6	104.0
1933	10.5	77	46	8.7	7.6	2.3	.4	5.3	2.2	53.1	.3	.2	1.7	74.6	70.3	12.4	84.7	62.2	203.2	185.3	81.4	71.3	70.7	16.4	98.7
1932	10.1	67	38	9.2	11.0	2.1	.5	5.0	4.8	29.4	.3	.4	1.5	77.2	59.0	11.6	79.1	60.4	195.5	180.3	87.2	73.4	65.4	13.3	99.5
Maryland:																									
1935	13.5	64	30	5.4	1.1	2.2	2.6	3.8	1.2	25.0	(1)	1.1	4.9	86.3	129.2	27.5	143.0	118.4	321.2	297.0	150.8	138.2	67.1	7.9	141.4
1934	13.3	69	32	6.0	1.7	16.1	2.3	9.4	1.2	11.2	.4	.8	.1	81.3	124.2	25.5	134.9	108.3	318.1	289.8	144.6	130.2	57.2	6.8	152.9
1933	12.9	62	27	5.2	.7	.2	2.9	2.4	1.1	26.5	(1)	1.0	1.0	87.8	118.3	27.2	132.6	103.0	308.9	278.3	126.8	115.6	61.6	9.1	154.2
1932	13.3	70	36	4.7	1.6	2.0	2.0	7.8	4.0	29.0	.2	.6	1.1	95.7	116.4	27.8	145.2	111.8	304.4	278.1	140.2	114.1	71.4	13.8	149.5
Michigan:																									
1935	10.6	56	22	5.4	.5	6.8	2.9	2.7	.8	21.6	(1)	.2	1.0	42.7	102.7	24.9	112.6	86.3	278.0	253.9	111.9	98.9	63.6	4.0	62.0
1934	10.7	57	22	5.8	.8	1.0	5.7	3.4	1.0	16.5	.2	.2	.6	46.0	101.4	23.6	119.3	91.2	278.0	252.7	106.5	92.7	67.4	5.0	64.5
1933	10.2	52	17	5.9	.6	4.1	4.6	3.7	2.3	28.1	.1	.4	.8	51.1	97.2	23.4	114.3	86.1	266.4	240.9	75.8	64.2	63.5	4.8	63.1
1932	10.3	57	22	6.3	.6	5.8	3.0	3.8	2.6	31.5	.3	.5	2.0	52.0	2.4	23.4	115.9	88.4	254.9	230.9	91.0	77.6	63.8	4.7	61.7
Minnesota:																									
1935	10.5	50	20	5.6	.2	4.2	4.8	3.4	.5	24.4	.1	1.0	2.1	37.0	131.0	23.6	108.5	86.1	244.8	220.5	109.5	98.4	65.3	3.2	50.4
1934	10.6	49	18	5.9	.8	2.5	1.5	4.5	1.0	20.5	.3	1.4	1.0	39.3	130.1	24.1	107.7	84.7	252.6	227.7	110.8	96.5	96.6	2.7	56.1
1933	10.2	53	23	5.1	.6	4.8	2.2	3.0	1.1	41.0	.2	1.2	1.8	43.2	131.6	21.5	100.5	82.1	232.8	209.8	82.7	72.5	62.0	4.5	58.4
1932	9.8	47	15	4.3	.3	.2	2.7	1.2	.5	36.8	.3	.7	1.2	43.1	120.4	20.6	101.8	78.0	216.5	197.4	82.5	73.3	60.6	2.4	56.8
Mississippi: [4]																									
1935	10.0	(1)	(1)	(1)	1.4	1.8	.4	6.0	3.4	76.0	.2	.1	1.2	52.0	53.9	11.6	73.4	59.6	120.7	105.9	77.8	83.7	50.7	9.1	91.8
1934	10.2	(1)	(1)	(1)	1.8	29.0	.4	15.4	2.4	38.9	.7	.6	.4	58.1	48.9	8.3	80.5	72.2	115.4	99.3	94.8	92.6	46.8	10.8	74.3
1933	9.5	(1)	(1)	(1)	3.8	4.1	.2	8.0	2.7	61.6	.2	.4	1.4	62.2	47.9	6.8	74.2	64.4	110.2	98.1	67.8	65.9	40.1	9.3	60.8
1932	9.2	(1)	(1)	(1)	2.5	(1)	.5	5.6	5.6	40.3	.8	.1	1.3	73.9	47.4	5.3	73.7	66.8	94.7	82.2	67.3	63.2	41.7	7.9	73.8

[1] Data not available.

[2] No deaths.

[4] 5 months only.

Mortality from certain causes in the first 6 months of 1935, with comparative data for the corresponding period in preceding years—Continued

State and period	All causes, rate per 1,000 population (annual basis)	Rate per 1,000 live births: Total infant mortality	Rate per 1,000 live births: All except malformations and early infancy	Rate per 1,000 live births: Maternal mortality	Death rate per 100,000 population (annual basis): Typhoid fever (1, 2)	Measles (7)	Scarlet fever (8)	Whooping cough (9)	Diphtheria (10)	Influenza (11)	Poliomyelitis (16)	Lethargic encephalitis (17)	Meningococcus meningitis (18)	Tuberculosis, all forms (23–32)	Cancer, all forms (45–53)	Diabetes (59)	Diseases of the nervous system (78–89)	Cerebral hemorrhage, apoplexy (82a, b)	Diseases of the circulatory system (90–103)	Diseases of the heart (90–95)	Diseases of the respiratory system (104–114)	Pneumonia, all forms (107–109)	Diseases of the digestive system (115–129)	Diarrhea and enteritis, under 2 years (119)	Nephritis (130–132)
JANUARY TO JUNE—con.																									
Montana:																									
1935	12.1	63	(2)	5.2	1.9	17.3	3.0	6.0	3.4	59.3	.4	1.1	3.0	45.4	100.6	18.4	109.3	83.7	225.7	208.8	170.1	155.1	104.4	6.4	79.2
1934	10.3	49	(2)	6.5	1.5	2.6	3.4	3.4	1.9	37.9	(2)	1.1	1.5	49.2	89.0	20.3	101.4	76.6	194.1	177.2	107.8	92.4	80.7	6.4	72.5
1933	9.7	59	(2)	5.2	2.9	4.4	2.9	4.4	2.2	60.6	(2)	1.8	.4	54.4	79.7	12.1	99.9	76.8	169.8	158.7	83.8	71.3	69.1	4.8	62.1
1932	9.9	49	(2)	5.3	2.2	3.0	1.5	6.0	.7	56.8	1.5	1.1	1.9	55.7	86.3	19.1	106.5	73.6	183.5	165.2	85.6	69.5	68.4	3.4	68.8
Nebraska:																									
1935	10.1	48	24	5.8	.6	11.3	3.5	1.2	1.4	35.1	.3	.6	4.2	22.2	100.0	20.1	132.0	103.3	211.8	194.0	127.4	111.6	66.6	2.9	49.3
1934	9.8	48	18	6.2	.4	2.3	1.7	8.5	1.2	21.7	.4	.6	1.2	24.7	111.4	20.7	124.5	99.1	205.8	185.6	103.6	92.0	69.8	3.0	59.2
1933	9.9	59	26	4.5	.3	1.0	2.0	1.6	1.3	56.6	.1	.4	.9	25.9	102.8	16.8	129.0	103.1	217.5	190.2	103.1	91.1	57.2	2.9	61.6
1932	9.4	45	16	5.4	1.3	.3	2.4	1.9	1.6	42.0	.3	.4	.7	22.5	102.1	23.8	116.0	95.3	196.1	174.3	90.6	69.7	62.2	4.0	82.9
New Jersey:																									
1935	10.6	51	(2)	4.8	.5	2.5	1.1	2.8	1.2	14.8	.3	.6	.8	52.4	119.2	30.0	100.3	80.6	325.0	302.9	91.0	81.0	56.0	3.3	86.4
1934	11.0	53	(2)	5.2	.4	2.0	2.4	1.6	1.4	8.1	.2	.9	.3	56.2	121.1	29.0	107.1	87.7	331.7	309.9	97.6	85.9	60.1	5.1	90.7
1933	10.8	50	(2)	5.4	.8	2.9	2.3	.7	.7	18.6	.1	.7	.6	60.0	115.8	30.2	110.6	86.3	303.5	280.8	98.3	86.9	62.7	4.8	92.2
1932	10.6	52	(2)	6.3	.3	1.5	2.1	4.2	2.6	22.2	.4	.7	.9	65.8	105.9	26.8	107.1	81.9	280.7	248.6	98.7	83.5	58.6	4.4	95.6
New York:																									
1935	12.1	54	23	5.7	.4	2.4	2.2	2.9	.9	10.4	.2	.6	2.7	60.1	140.8	34.3	101.3	78.8	370.2	336.9	119.5	108.4	68.1	6.5	86.2
1934	12.4	58	25	6.0	.4	1.0	2.0	1.5	1.2	9.1	.5	.5	.8	62.6	137.5	33.7	76.8	52.6	400.6	343.6	125.9	113.5	71.9	7.1	91.4
1933	11.9	58	26	7.1	.4	4.6	2.5	2.6	1.3	16.5	.2	.9	.9	63.2	126.2	32.6	82.7	54.5	364.5	308.3	123.3	111.9	69.4	6.5	78.5
1932	12.2	58	25	6.4	.5	2.7	4.5	2.4	2.9	18.4	.3	.8	1.7	69.6	125.5	33.6	81.0	55.0	369.6	314.6	136.7	124.6	66.9	5.7	81.1
North Carolina:																									
1935	10.9	72	(2)	6.9	1.1	4.0	.6	12.7	3.2	49.5	1.5	.4	1.2	62.4	51.3	10.7	(2)	(2)	(2)	(2)	(2)	121.4	(2)	16.8	(2)
1934	11.3	83	(2)	7.5	.7	16.8	1.6	13.9	4.5	31.2	.4	.3	.7	68.6	52.2	11.5	(2)	(2)	(2)	(2)	(2)	137.4	(2)	17.2	(2)
1933	9.8	73	(2)	6.7	2.1	3.6	.9	5.9	2.7	48.4	.2	.4	.2	68.3	48.6	10.7	(2)	(2)	(2)	(2)	(2)	79.6	(2)	20.3	(2)
1932	9.8	71	(2)	7.5	3.4	2.6	1.0	8.6	3.5	27.2	.5	.9	.8	67.0	44.3	11.6	(2)	(2)	(2)	(2)	(2)	104.3	(2)	15.5	(2)

Ohio:																								
1935	12.5	57	25	6.8	.6	3.6	4.5	3.8	2.8	36.4	.4	1.1	4.6	56.2	118.6	26.9	139.5	117.5	296.0	265.0	107.0	97.2	61.3	3.4	83.7
1934	12.1	60	26	6.6	1.0	3.9	4.3	5.8	2.1	23.4	.1	.7	1.2	56.5	115.9	26.1	141.2	118.1	300.2	267.7	115.0	103.0	63.4	3.5	88.2
1933	11.2	53	19	6.7	1.0	3.1	3.6	2.1	1.9	35.3	.3	1.2	.6	56.4	112.5	23.5	130.4	109.8	286.6	254.8	78.1	68.4	60.7	3.7	81.9
1932	11.7	60	25	5.8	.8	4.2	4.1	7.0	3.5	41.5	.3	.8	1.2	64.0	109.9	26.8	139.0	116.6	287.5	252.2	97.5	92.2	58.5	4.6	83.1
Pennsylvania:																								
1935	11.7	58	29	5.4	.6	4.3	2.8	2.5	1.7	28.9	.2	.9	1.4	49.1	109.0	29.3	114.9	87.7	321.6	297.5	123.4	107.8	56.2	5.2	92.7
1934	11.9	67	35	6.5	.8	4.9	3.4	3.8	2.5	20.9	.2	.6	.7	52.0	105.9	30.8	114.7	89.2	322.4	289.8	123.5	108.0	61.2	7.0	100.6
1933	11.3	57	26	5.4	.5	1.7	4.1	1.7	2.7	37.0	.4	.7	1.2	53.5	103.4	28.6	111.9	88.5	286.9	260.7	93.4	80.7	58.5	7.0	101.1
1932	11.7	62	30	6.5	1.1	4.0	3.7	5.6	4.8	39.8	.4	.7	1.6	57.3	102.6	26.8	118.7	92.0	293.8	258.1	115.7	101.9	62.8	8.2	101.1
Rhode Island:																								
1935	11.9	55	20	2.9	.3	1.7	.6	1.7	.6	12.5	.3	.3	4.0	53.0	139.0	37.3	119.1	98.0	377.7	353.2	109.4	99.7	65.8	6.0	102.3
1934	12.0	60	23	6.1	.9	(1)	.9	4.3	(1)	10.9	.3	.3	.6	46.0	136.2	33.4	124.3	100.3	354.5	329.3	112.5	100.5	61.7	4.6	120.5
1933	12.0	60	20	5.0	.3	(1)	2.0	2.9	.6	31.6	(1)	.6	(1)	54.3	135.9	36.5	125.8	102.6	317.4	292.4	103.7	94.8	66.9	4.6	127.0
1932	12.7	65	29	5.2	(1)	11.8	6.6	1.7	6.6	18.2	.6	.9	(1)	58.9	143.9	37.6	131.5	108.3	311.4	286.6	148.8	136.9	70.2	4.6	125.1
South Carolina:																								
1935	11.5	90	(1)	10.6	6.1	1.8	.2	11.5	1.5	73.7	.8	1.0	1.8	53.3	45.7	11.1	112.4	99.9	204.9	191.7	116.7	109.5	33.5	8.2	97.1
1934	11.6	104	(1)	11.1	3.7	17.4	.5	18.3	3.0	53.9	.7	2.3	.7	56.5	46.9	12.3	(1)	(1)	204.0	(1)	(1)	120.8	33.6	(1)	109.1
1933	9.6	64	31	4.3	8.3	1.1	2.9	4.6	2.3	7.6	.6	.3	.3	45.0	83.2	22.9	107.9	81.5	191.6	159.8	89.8	79.7	49.3	4.6	58.8
1932	8.2	53	24	4.2	2.0	(1)	1.4	5.2	3.4	37.9	1.1	.3	.9	41.4	83.6	16.1	84.2	67.2	179.0	158.9	63.5	50.8	59.2	4.3	39.6
South Dakota: [4]																								
1935	9.9	62	33	8.0	.7	9.2	2.1	4.4	1.4	55.7	1.4	.7	.3	41.4	90.6	20.9	107.4	81.0	158.3	138.1	156.6	139.2	54.4	5.8	61.5
1934	10.0	66	31	6.1	1.0	30.2	2.7	9.9	1.0	40.5	.7	.3	(1)	35.0	88.8	23.0	109.4	83.3	171.1	149.8	120.7	110.1	60.0	5.5	60.0
1933	10.1	68	33	4.2	10.0	.7	3.1	4.8	2.4	80.2	.7	.3	.3	46.8	87.1	23.8	107.4	83.3	199.4	166.7	102.3	91.6	52.3	5.5	62.0
1932	8.3	53	24	4.3	2.4	(1)	1.4	5.8	3.1	42.0	1.4	(1)	.7	41.3	84.6	16.2	85.7	68.5	184.4	166.1	66.4	52.3	60.9	4.5	40.2
Tennessee:																								
1935	10.8	69	42	7.7	2.8	2.3	.5	22.9	3.9	62.5	.6	.6	5.0	91.4	66.1	12.1	103.9	81.4	160.1	145.9	142.8	121.4	67.6	11.9	63.7
1934	10.9	77	47	6.6	3.0	30.1	1.5	8.4	3.1	47.6	1.0	1.1	1.4	88.8	62.4	11.6	100.6	80.0	162.2	150.0	130.8	109.2	73.4	14.9	64.1
1933	10.2	73	47	6.1	4.3	2.3	1.1	6.1	2.7	62.0	.7	1.1	.8	92.6	56.7	10.7	86.7	59.8	116.1	99.2	98.0	85.8	72.3	14.4	58.9
1932	10.6	70	43	7.0	5.2	.2	.6	7.5	3.5	59.3	.4	.8	1.9	98.8	54.1	10.7	89.8	61.5	118.2	103.5	104.0	95.1	70.7	14.2	68.9
Virginia:																								
1935	12.6	71	(1)	5.8	1.1	10.5	1.2	11.9	3.8	62.9	.4	.6	5.3	80.0	75.9	17.3	133.0	105.5	267.5	247.0	123.7	112.7	54.7	7.7	97.0
1934	12.3	72	(1)	7.0	2.1	9.9	1.4	7.5	3.9	38.2	.5	.7	1.6	81.0	73.3	19.4	127.5	100.2	258.0	239.9	115.1	102.1	54.2	9.4	97.8
1933	11.5	71	(1)	6.1	2.5	3.6	1.7	4.7	2.4	63.1	.2	.5	1.2	84.6	73.1	15.4	130.5	104.1	217.9	199.8	93.1	82.6	59.0	12.9	92.8
1932	11.5	70	(1)	6.9	2.7	.9	1.2	17.3	5.1	49.8	.7	.8	1.6	90.4	67.9	15.7	117.5	94.0	207.6	191.9	96.9	85.3	60.1	11.7	98.7
Washington:																								
1935	11.4	45	15	5.8	.4	2.1	1.7	2.9	1.1	23.3	.9	2.0	2.4	51.6	132.9	23.9	124.9	102.3	306.2	279.0	69.6	59.9	64.5	2.1	82.1
1934	10.8	(1)	(1)	(1)	1.8	1.4	1.1	3.6	.6	14.3	.9	2.0	1.4	49.3	129.3	20.9	117.6	91.8	292.7	260.1	71.9	62.0	57.2	1.6	75.1
West Virginia																								
1935	10.2	65	32	6.4	3.1	11.9	4.9	11.2	5.8	44.4	.3	.6	4.4	61.9	71.3	12.1	93.5	72.1	161.5	145.4	118.3	109.0	59.3	5.9	70.5
1934	9.7	69	30	6.3	1.8	3.4	3.8	10.6	4.5	36.1	.6	.5	1.8	54.3	64.5	10.6	102.9	82.4	147.1	126.1	110.3	99.1	47.1	7.8	65.5
1933	9.2	(1)	(1)	5.2	3.3	4.1	1.6	5.2	4.0	55.8	1.3	.2	1.1	56.0	66.7	10.5	103.6	72.6	126.4	115.0	84.1	73.0	61.5	15.7	80.1
1932	9.6	(1)	(1)	5.6	4.1	16.5	2.4	13.7	8.6	52.3	.7	.4	1.3	56.0	61.9	13.2	109.1	80.5	122.3	109.7	100.0	88.9	64.2	19.5	63.9
Wisconsin:																								
1935	10.4	54	21	4.1	.1	3.9	5.2	1.5	.5	29.5	.1	1.1	1.7	37.3	123.3	24.8	(1)	88.3	(1)	255.5	(1)	77.5	(1)	4.7	70.9
1934	10.5	56	22	4.3	.3	3.7	1.7	4.2	1.0	15.8	.3	.3	.6	40.1	122.2	24.7	(1)	88.8	(1)	243.7	(1)	87.4	(1)	5.0	71.5
1933	10.3	56	21	5.6	.5	1.5	1.6	1.6	.5	43.0	.3	.5	.7	44.0	108.7	27.0	(1)	92.5	(1)	237.1	(1)	65.8	(1)	6.5	65.9
1932	10.3	55	21	4.5	.3	2.7	2.3	2.7	1.9	36.1	.5	1.1	1.1	49.4	117.9	23.7	(1)	91.4	(1)	223.7	(1)	78.8	(1)	5.9	69.2

[1] Data not available.

[2] No deaths.

[4] 5 months only.

RAT LEPROSY

Observations Concerning Transmission of the Infection through the Nose

By N. E. Wayson, *Senior Surgeon, U. S. Public Health Service,* and Eichi Masunaga, B. S., *U. S. Leprosy Investigation Station, Honolulu*

The portal of entry through which the micro-organism of the leprosy-like disease of wild rats enters the tissues has not been determined. It has been suggested that it is probably introduced through small wounds in the skin caused by trauma or by the bites of insects. The skin of rats is particularly subject to minor injuries or abrasions produced by biting, scratching, or the plucking of hairs, and it is assumed that the micro-organism may be introduced into these lesions by soiling them with material containing the inoculum. This is thought to be accomplished through the soiling of the teeth or the claws which inflict the wounds, or by bodily contact with the open lesions of infected animals. This belief is perhaps supported to a slight degree by the fact that the infection can occasionally be accomplished experimentally by abrading the skin and rubbing suspensions of the bacteria into it. However, it is to be noted that these suspensions must contain very large numbers of bacteria in order to obtain even a small percentage of infections by this method.

The production of the disease by the introduction of the inoculum through the bites of the ectoparasites common to rats has been attempted by several investigators, including Marchoux (*1*) and Markianos (*2*), who have concluded that the infection is not transmitted in this manner. Wherry (*3*), Currie and Hollmann (*4*), and Markianos (*2*) have all reported that they have found acid-fast bacteria resembling the organism of rat leprosy in suspensions made of the crushed bodies of such parasites which had fed on infected animals, but the subcutaneous introduction of such suspensions by Markianos did not produce the infection.

The possibility of the entrance of the organism through the mucous membrane of the nasopharynx seems to have had less consideration. The bacterium has been found to be commonly present in the noses of leprous wild rats in San Francisco and among white rats which were experimentally infected by subcutaneous injections.[1]

[1] The strain of rat leprosy used throughout these experiments was developed in white rats by direct transfer from leprous wild rats in San Francisco. Its identity has been repeatedly checked during this experimentation by its morphology and tinctorial qualities, and by its failure to grow on artificial media and in guinea pigs, as well as by the microscopic and histological changes produced by it in rats.

TABLE 1.—*Number of rats in which acid-fast bacteria were found in the nose*

LEPROUS RATS

	Wild rats Natural infection			White rats Experimental infection (subcutaneous inoculation)		
	Total examined	Nose positive	Nose negative	Total examined	Nose positive	Nose negative
Skin lesions with ulcerations	24	20	4	0	0	0
Skin or lymph node lesions without ulcerations	32	22	10	58	48	10
No lesions;[1] bacteria in lymph nodes or skin	3	0	3	0	0	0
Total	59	42	17	58	48	10

NONLEPROUS RATS (CONTROLS)

	Wild rats[2]			White rats		
	Total examined	Nose positive	Nose negative	Total examined	Nose positive	Nose negative
No lesions;[1] no bacteria in lymph nodes or skin	55	3	52	70	0	70

"Positive" indicates that acid-fasts were found.
"Negative" indicates that acid-fasts were not found.

[1] The axillary and inguinal lymph nodes of the wild rats were those which were regularly examined. Attention was directed to the cervical nodes in the experimental studies at a later date.
[2] These rats came from the same or nearby localities in which the leprous rats were caught.

While there is a likelihood that the wild rat may infect his nose by contact with ulcerated lesions, it is improbable that the subcutaneously inoculated animal does so. It is also unlikely that the inoculated rats had infected their noses previous to the inoculation, since both the test animals and the controls were selected at random from the clean supply stock. Since the organism appears to be so frequently present in the nose, it may be that its dissemination from this site is a natural phase of the life cycle of the parasite. Its introduction into the nose of a new host can be accomplished either through the nosing and licking which goes on between rats, or perhaps through droplet infection, and minute wounds of the skin could thus be readily soiled subsequently. This has been shown by finding acid-fast bacteria resembling that of rat leprosy in the noses of 54 of 75 normal rats kept in small groups in contact with experimentally infected rats in boxes for periods of 3 to 8 months (fig. 1). It seems very improbable that these findings are accidental, since similar bacteria were not found in the noses of 70 control rats which had not had such contacts (table 1). Furthermore, it was observed that nurslings show positive findings after a shorter period of exposure than occurs with older animals, and in general a longer period of exposure of all ages results in a greater number of positive findings. The possibility of spreading the bacterium by indirect methods is

suggested by the findings among groups of normal animals which were kept in cages about 4 inches distant from those containing animals with ulcerated lesions about the face for periods of 3 to 5 months. Acid-fast bacteria were found in 30 of 44 of the exposed groups which were examined.

Among the rats which were held in contact with experimentally infected rats in boxes, 8 were held for 8 months, and their examination included preparations from their cervical lymph nodes. Acid-fast bacteria were found in the cervical nodes of 3 of this group. In 1 of these 3, one node was swollen and adherent to the surrounding structures. Microscopic section of this node revealed a typical rat leprosy granuloma containing large numbers of acid-fast bacteria (fig. 2). These findings suggested the probability of infecting rats by placing the inoculum on the nasal mucous membrane.

During the course of 3 years a number of experiments have been made in which a few drops of a suspension of rat leproma have been instilled once or twice into the nostrils of each of a group of rats without traumatising the mucous membrane. Ninety-four rats which were inoculated in this manner were subsequently examined. Acid-fast bacteria were found in the cervical lymph nodes in 69 of them (fig. 3). Among 66 which were thus treated and examined 3 or more months later, 58 showed positive findings in the cervical nodes. The larger percentage of negative findings occurred in those groups which were examined after less than 3 months.

Among 1 lot of 8 of the 66, the examinations and findings were as follows: One, 3 months later, with bacteria in mediastinal nodes; four, 7 months later, with bacteria in the cervical nodes; one, 9 months later, with bacteria in the nodes and skin without macroscopic lesions in the skin; one, 11 months later, with bacteria and macroscopic lesions in the skin; one, 15 months later with bacteria in macroscopic lesions generally scattered through the skin and viscera (fig. 4).

In each of two other instillation experiments the bacteria were found in locations remote from the nose. In one animal the bacteria were in mesenteric nodes, and in another they were in lepromata, which developed in the cervical node, in the adjoining neck tissues, and in the lung.

The animals used in these instillation experiments were kept in boxes or cages in which there were no infected animals, but precautions were not taken to avoid all indirect contact through ectoparasites. Examinations were made, however, of other superficial lymph nodes, "axillary" and "inguinal", of 29 of those in which positive findings in the cervical nodes were obtained after 3 or 4 months had elapsed since the nasal instillations. Acid-fasts were not found in any.

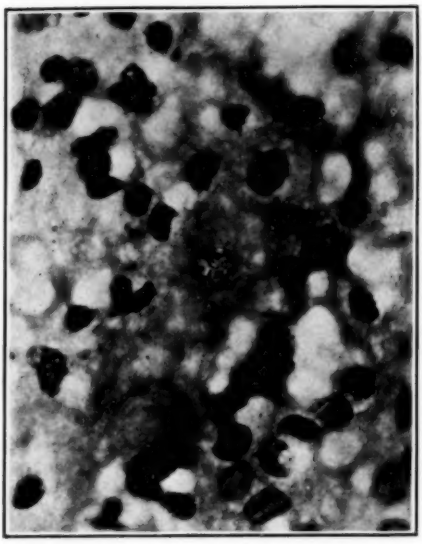

FIGURE 1.—Cluster of acid-fast bacteria in a smear from the nasal mucous membrane of a contact rat.

FIGURE 2 *(a)*.

FIGURE 2 *(b)*.

FIGURE 2 *(c)*.

Figures 2 *(a)*, *(b)*, and *(c)* show three magnifications of a leproma in a cervical lymph node of a contact rat. Figure 2 *(c)* shows the bacteria in one of the nests of epithelial cells *(b)* of the granuloma.

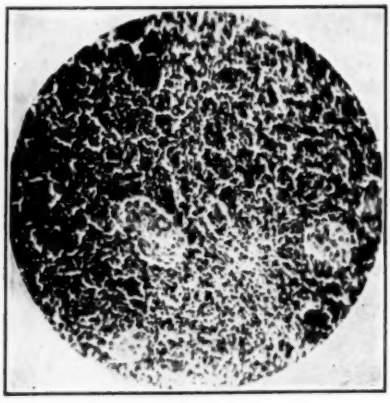

FIGURE 3 *(a)*.

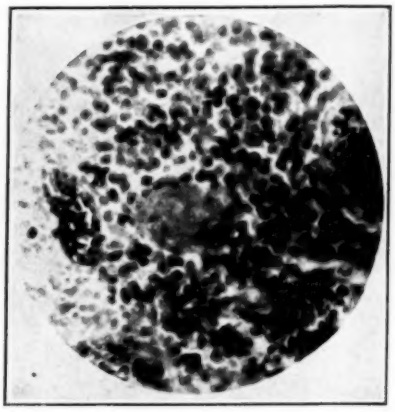

FIGURE 3 *(b)*.

Microscopic lesions *(a)* and bacteria *(b)* in a cell of the lesion in the cervical lymph node of a rat into whose nose a suspension of the rat leproma had been instilled. The size of this node was but little, if any, larger than normal, but the node was of firmer consistency.

FIGURE 4 *(a)*.—A portion of the skin of a rat which was infected through the nose. Note the appearance, which is characteristic of natural infections.

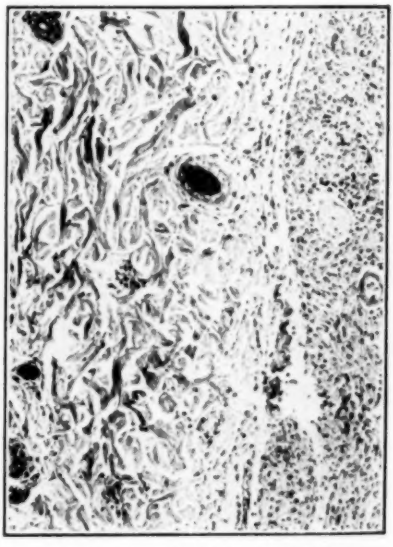

FIGURE 4 *(b)*.—A section of the skin of *(a)*, showing the infiltration typical of rat leprosy.

A final experiment was made in which each of 22 rats were searched for ectoparasites, dusted with pyrethrum, treated by instillation, and held segregated for 3 months as one rat to a box or compartment in a room separated from other experimental work. Dusting with pyrethrum was repeated several times during the 3 months, and but 2 to 4 mites were found on any rat when they were killed for examination. The cervical lymph nodes of 19 of these rats contained acid-fast bacteria when examined at the end of the 3 months' period, but no acid-fasts were found in the axillary or inguinal nodes of any of them, nor were any found in the cervical nodes of 16 uninoculated control rats held in groups in boxes in the same room throughout the period.

CONCLUSIONS

Klingmueller (*5*) states that no one has shown that the bacillus of rat leprosy will traverse the normal nasal mucous membrane of the rat. The observations and findings made in these experiments seem to justify the conclusion that the bacterium will pass through the uninjured nasopharyngeal mucous membrane which is normal from that standpoint. Acid-fasts were commonly found in the noses of rats infected with rat leprosy; and under the conditions of these experiments, the bacterium was transmitted to the noses of normal rats either by direct or by indirect contact with diseased rats, and the disease developed subsequent to a deposit of the inoculum on the mucous membrane without trauma to it.

DISCUSSION

The frequency with which infection through the nose occurs in nature among wild rats is unknown. However, the cervical lymph nodes are not commonly the site of macroscopic changes in such infections, whereas the axillary or inguinal nodes are frequently enlarged or exhibit some change. These latter findings would suggest a portal of entry other than the nose. On the other hand the bacteria and microscopic lesions are often found in lymph nodes which do not present gross changes, and it seems highly probable that the microorganisms may pass from such nodes to the circulation without producing macroscopic changes in them. Further observations of microscopic preparations of the cervical lymph nodes of wild rats should be of assistance in determining the frequency with which they are involved, and may thus indicate the frequency with which the infection enters through the nose.

No conclusions are offered from these findings in a leprosy-like disease in rats concerning the processes of leprosy in man. Nevertheless, it seems proper to point out analogous conditions and obser-

vations in the two diseases, since consideration of the similarities which occur in some other diseases of animals has contributed to the knowledge of analogous diseases of man. Both diseases are apparently caused by acid-fast bacteria of comparable morphology, which have not been consistently cultivated in artificial media or in more than one species of host; both diseases are characterized by changes in the blood vessels, the formation of granulomatous infiltrations, extension along contiguous lymph spaces, and by the invasion of the lymph and blood streams by the respective bacteria which may produce granulomatous changes in many localities or tissues of the body, but with a marked selectivity for those of the skin and superficial lymph nodes. The respective bacteria can be demonstrated in or on the nasal mucous membrane in most cases of each disease and in some individual persons or rats in which macroscopic or clinical evidence of disease is not detected. Horcicka, Kitasato, L. Glueck, Roemer, Plumert, Serra, and Zechmeister (*6*) are all quoted by Klingmueller as having reported the presence of acid-fast bacteria in the noses of those who are in frequent intimate contact with leprous persons. Sorel and Leboeuf (*7*) are quoted by Klingmueller, and Marchoux (*8*) is quoted by Jeanselme as having discovered acid-fasts in the lymph nodes of attendants to leprous persons, thus suggesting the invasion of the tissues of apparently well people by the bacterium of leprosy. It will be noted that similar observations have been made in rats in these experiments on the leprosy-like disease.

Jeanselme (*9*) states in a recent publication that no leprologist believes any longer that the habitual route of infection with leprosy is through the nose. He does not cite investigations in substantiation of their viewpoint, and it is believed that further studies should be conducted before this hypothesis is discarded. This latter opinion seems especially pertinent, since the suggestions that different areas of the skin afford all portals of entry are not well supported by clinical or epidemiological experience.

SUMMARY

1. Acid-fast bacteria have been found frequently in the noses of wild rats infected with the leprosy-like disease of rats, and likewise in white rats experimentally infected by subcutaneous inoculation.

2. Normal white rats in prolonged direct or indirect contact with infected rats harbored acid-fast bacteria in the nose and developed the disease under the conditions of the experiments.

3. Rat leprosy has developed in white rats subsequent to the instillation of a suspension of rat leproma into the nostril without trauma to the nasal mucous membranes.

REFERENCES

(1) Marchoux, E.: Troisème Conf. Internat. d. la Lèpre, pp. 111–113. (1924.)
(2) Markianos, J., quoted by Klingmueller, Victor: Die Lepra, p. 139. Berlin. Julius Springer. (1930.)
(3) Wherry, Wm. B.: Rat lepra bacilli in the rat louse. Jour. Infect. Dis., Vol. 6, p. 633. (1909.)
(4) Currie, D. H., and Hollmann, H. T.: A contribution to the study of rat leprosy. Pub. Health Bull. No. 41, pp. 18–31. (November 1910.)
(5) Klingmueller, Victor: Die Lepra, p. 138. Berlin. Julius Springer. (1930.)
(6) Horcicka, Kitasato, L. Glueck, Roemer, Plumert, Serra, and Zechmeister, quoted by Klingmueller, Victor: Die Lepra, p. 226. Berlin. Julius Springer. (1930.)
(7) Sorel and Leboeuf, quoted by Klingmueller, Victor: Die Lepra, p. 226. Berlin. Julius Springer. (1930.)
(8) Marchoux, E., quoted by Jeanselme, E.: Biologie Médicale, Vol. 24, p. 463. (October 1934.)
(9) Jeanselme, E.: De la lutte contre la lèpre a l'époque contemporaine. Biologie Médicale, Vol. 24, p. 477. (October 1934.)

STUDIES OF SEWAGE PURIFICATION

III. The Clarification of Sewage—A Review [1]

By Emery J. Theriault, *Principal Chemist, United States Public Health Service*

It is frequently remarked that present-day methods of sewage treatment have greatly outstripped the development of rational theories of plant behavior. This situation, of course, is by no means unique in the general field of practical applications. In sewage treatment, nevertheless, the rationalization of plant procedures has doubtlessly been greatly retarded by the (at times) hopeless outlook of the fundamental problem regarding the quantitative description of reactions between the oxygen-consuming impurities of sewage, at a concentration of approximately 0.001 M, and the various kinds of "activated sludges", also known as "biological slimes" and more accurately described as zoogleal aggregates.

With such unpromising material at hand, it is to be expected that the theories in vogue are generally founded on more or less pertinent analogies with more amenable systems rather than on the direct examination of sewage colloids or bio-colloids. Sustained research, for example, on the various theories of adsorption as a mechanism for the removal of organic matters from sewage by sludges becomes impossible when no absorption is observed, using sludge drawn from an activated sludge plant. Likewise, the evaluation of various theories of colloidal precipitation is a discouraging task when the sewage colloids refuse to agglomerate or when the agglomerated solids decline to settle. The development of biological theories is greatly hampered by unsurmounted difficulties in the matter of cultivating protozoa and allied organisms in pure culture; bacterial counts may fail to yield more than 1 percent of the organisms present

[1] This is the first of a series of papers summarizing the studies of adsorption by activated sludge conducted at the stream pollution investigations station of the United States Public Health Service, Cincinnati, Ohio.

in the sludge. Theories of enzymatic action have frequently been advanced, but in no case have the hypothetical clotting, lytic or oxidative agents been produced.

In another sense, the present unsatisfactory state of the sewage problem is closely related to the controversy, as reviewed by Wilson (1930), "over the imagined respective functions of the biological and physical agencies." No single theory has been proposed which will account satisfactorily for all of the diverse phenomena of sewage treatment. In general, the physical or colloidal theories have explained the purification of sewage either by the coagulation of the sewage colloids in various ways or else by the absorption of the organic matters on the sludge, to the frequent neglect of the subsequent biological oxidation of the coagulated or absorbed impurities. On the other hand, this preliminary process of clarification has often been ignored or even denied by the proponents of the biological theories.

It is to be surmised that the various theories of sewage purification do not all possess an equal degree of credibility. Nevertheless it is safe to assume that biological oxidation in some form must necessarily be considered in any general theory of sewage purification by the usual processes. Greater uncertainty exists as to the validity of the clarification concept. For the purposes of the present discussion, leading to the presentation of a new theory of sewage clarification, the review of the scattered literature will therefore be devoted largely to the phenomenon of clarification as a process preliminary to and distinctly separated from the subsequent process of biological oxidation.

EARLIER THEORIES

The earliest recorded scientific investigation of sewage treatment is probably that of Frankland (1868) with the British Rivers Pollution Board. Working under laboratory conditions, with glass cylinders, Frankland gave the first clear-cut demonstration of the role of atmospheric oxygen in the purification of sewage and laid the foundation for the principle of "intermittent filtration." Frankland assumed that the dissolved organic matters of sewage were oxidized by the oxygen of the air as the sewage dripped through a filter or a porous soil.

With the recognition of the biological character of filter growths, the simple theory of direct oxidation advanced by Frankland became untenable. The numerous reports of the Lawrence Experiment Station (1887 et seq.) are to be consulted for the development of theories of direct biological oxidation of organic matters in the *slow* passage of the sewage through a filter. Commenting on these earlier observations Dunbar (1908, pp. 121–122) says: "If Frankland's experiments are to be regarded as the basis of modern biological methods of sewage treat-

ment, they have really attained this position with the aid of the systematic and scientifically conducted experiments of the Massachusetts State Board of Health."

Dunbar's classical experiments were conducted at the Hamburg Hygienic Institute (1897–1900). He considers that the oxidation of sewage matters in their slow passage through a filter was a possible explanation "* * * so long as it could be maintained that sewage remained about 3 days in the filter; but when it was shown that the sewage left a 3-foot filter thoroughly purified within 10 minutes * * * the above explanation fell to the ground. It cannot be assumed that micro-organisms decompose highly complex molecules of organic substances within a few minutes or seconds * * *. It can only be assumed that the dissolved organic matters are first separated from the sewage during its passage through the filter, and are retained in the filter to be decomposed and oxidized by the micro-organisms during the succeeding period of rest." After examining theories of mechanical retention and of chemical combination, Dunbar concludes that "an explanation of the purification process must be sought in absorption phenomena."

The general theory of filter action proposed by Dunbar, and systematized by Ardern (1921), was somewhat as follows:

1. Coarse particles are transferred to the filtering media by surface attraction.

2. Dissolved matters are retained by adsorption phenomena accelerated by the growth of a biological slime on the filter materials.

3. The absorbed materials are oxidized by biological action and also by oxygen "ozonized by the high pressures existing in the gelatinous film."

4. The absorption process is prevented from ceasing by the action of micro-organisms under aerobic conditions.

5. There is a residue of organic matter, highly resistant to bacterial action, which either accumulates in the filter or is discharged with the effluent.

Dunbar's concept of the action of sewage filters is therefore properly described not simply as an "absorption theory" but rather as a well-balanced summary of the then existing knowledge regarding sewage purification.

Stoddart's criticism of Dunbar's theory will serve as an illustration of objections brought to bear against absorption as a step preliminary to biological oxidation. "It has already been shown that the starting point is the assumption that micro-organisms cannot 'decompose highly complex molecular organic substances within a few minutes or seconds' * * * and with the exposure of the fallacious nature of this assumption, which may be characterized as an unwarrantable repression of scientific imagination, the theory itself falls to the

ground." (Stoddart, 1909, p. 201.) Stoddart's experiments controverting Dunbar's absorption theory were based on comparisons of rates of flow through filters of solutions of sodium chloride and of ammonium chloride. It will be shown in subsequent papers of this series that the absorption of ammonium chloride should be prevented by the sodium chloride, unless special precautions should be observed. It is also possible that the absorptive capacity of Stoddart's filter was exceeded. It must be admitted, nevertheless, that Dunbar's theory when first proposed was by no means free from objections, particularly in the matter of the removal from sewage of dialyzable or noncolloidal matters such as ammonia.

Biltz and Krohnke (1904), in their important paper on the first definite recognition of the colloidal nature of sewage, list three methods whereby the clarification of sewage may be accomplished. Chemical precipitation is represented by the use of ferric salts. Biological methods of sewage treatment are regarded as affording a means for the formation of absorption compounds between the colloids of sewage and the slimy, gelatinous coatings of the filter materials. These biological slimes, however, are regarded simply as colloid surfaces. The bacteria themselves, although identified with the biological slimes, are regarded as precipitants in reversed analogy to agglutination. The mechanical theory proposed by Biltz and Krohnke is referred by them to the absorption theory of Dunbar and earlier writers; they do claim, however, to have established a rational connection between the nature of the sewage and the methods of clarification. As to the fate of the absorbed materials, it was assumed that they might be oxidized by the action of "ferments" or else directly (unmittelbar) by atmospheric oxygen, "at all events essentially by purely chemical means." (*cf.* Jones and Travis, 1906, pp. 72 and 161.) This unsubstantiated part of the theory advanced by Biltz and Krohnke does not detract greatly from their demonstration that, ahead of corrections, at least one-third to one-half of the sewage matters are present as colloids.

Jones and Travis (1906), in their widely discussed paper on the clarification of sewage by filters at Hampton (England), advanced a somewhat different theory for the "de-solution" of sewage colloids. They maintain that the deposition of solids on the filter materials "is dependent upon the action of surfaces, as such", and not on the colloidal properties of the biological slimes. The efficiency of a filter should therefore be increased by the use of finely divided material. Jones and Travis (1906, p. 192, discussion) were afterwards willing to concede to Biltz and Krohnke, and also to H. W. Clark of the Lawrence Experiment Station, that "* * * colloids in gel form encouraged further deposition, and to this extent the bacterial coating of the (filter) material undoubtedly played a part."

Biochemical oxidation was practically disregarded by Jones and Travis (1906) and, as pointed out by Wilson (1930), could properly be omitted if their theory was restricted to sewage clarification. The claim that "the bacteria play only a subsidiary part in the purification" was, nevertheless, highly objectionable to proponents of biological theories. The view of bacterial purification held by Jones and Travis was clearly an expectancy of sludge liquefaction. "In Hampton, at the outset, absolute confidence was reposed in voracious capacity of the bacteria. Indeed, the announcement was made that the solids of sewage would all be 'eaten by the organisms'", thereby preventing the clogging of a relatively fine (0.5-inch) filter. Disillusionment in respect to the hydrolysis of the highly resistant sludge humus by micro-organisms is caustically expressed in the quoted statement, "The best organism I have at the sewage-works is the man with a barrow" (*cf.* Dunbar).

Although the so-called "Hampton doctrine" of Jones and Travis is frequently contrasted with the more general theory of Dunbar, it is clear that the views expressed by these workers, and also by Biltz and Krohnke, are in substantial agreement as to the mechanism for the primary removal of suspended or colloidal matters from sewage. In each case a purely physical theory is proposed, which does not account for the removal of matters in true solution, such as ammonia, sugar, etc. The Dunbar theory, however, does make suitable provision for the subsequent oxidation of the absorbed materials. The introduction of the time element, with its implication of preliminary absorption instead of direct oxidation by atmospheric oxygen or direct action by bacterial cells, must be regarded as Dunbar's greatest contribution to the sewage problem.

RATE OF CLARIFICATION

While it is plausible or even necessary to assume that some preliminary process of absorption must be operative in the clarification of sewage, it was by no means easy to separate this process from the accompanying process of biological oxidation when methods of sewage treatment were restricted to contact beds or trickling filters. The phenomenon of clarification may nevertheless be readily demonstrated by a suitable arrangement of parts in biological oxidizing devices utilizing "activated sludges" as absorbents. The experiments of Theriault and McNamee (1930) will serve as an example.

Using a biological device, designed to simulate a small stream with polluted water trickling over slime-covered stones, it was shown at the Stream Pollution Investigations Station of the United States Public Health Service that, starting with a grossly polluted water, the production of a highly clarified effluent could be effected in a period of

flow of about 20 minutes. The apparent reduction in the incoming pollution, neglecting absorption, was of the order of 90 percent. For polluted waters incubated under laboratory conditions, in the absence of deposited sludge, an equivalent degree of actual purification by biological oxidation could be accomplished only in 10 days, under comparable temperature conditions. The term "clarification" will be used in referring to the marked over-all improvement which results when sewage is treated for a brief period of time with certain biological slime growths, the so-called "activated" sludges. The term "purification" is properly restricted to the relatively slow but none the less effective process of biological oxidation. It is obviously desirable to distinguish clearly between these two methods whereby the pollutional characteristics of a sewage may be diminished.

Reference will again be made to these experiments later in this paper. Their particular virtue lies in the fact that the time element was very clearly defined and, also, that the tests were conducted on a semiplant scale in close approximation to natural conditions. It is reasonable to assume that the phenomenon of clarification demonstrated in these experiments is identical with the rapid improvement noted in trickling filters, contact beds, and similar devices where the control of experimental conditions offers many difficulties. In each case, however, it is certain that biologically active sludge is present and it can be assumed that the period of contact is brief, although divergent opinions on this particular point have been expressed.

Clarification, likewise, undoubtedly occurs in the activated sludge process, where the biological slimes are blown through the sewage instead of remaining adherent to stones as in processes of filtration. The time element in the activated sludge process can only be estimated with difficulty in an actual installation. Activated sludge, nevertheless, is admirably adapted to laboratory experimentation, and several workers have reported on its clarifying power under controlled conditions.

In the discussion following the paper by Young and Melling (1918, p. 19), Garfield refers to the experiments of Barraclough in which a reduction of about 50 percent in permanganate oxygen consumed, together with a decrease in free ammonia, was observed in absorption experiments where samples were taken "immediately" after mixing activated sludge with sewage. "The first point was that the activated sludge, upon being mixed with the sewage, immediately effected a purification which might be compared with the action of a very heavy chemical precipitant, * * *." Barraclough evidently was dealing with a very "good" sludge.

Cambier (1920) presented experiments indicating that the removal of the colloidal matters of sewage, together with the ammonia, is effected from the very start of the contact period with activated

sludge, although not at the first instant of contact as interpreted by Butler and Coste (1927). In work with fresh sludge approximately 50 percent of the ammonia was absorbed in 15 minutes, without any corresponding indication of nitrification.

Butler and Coste (1927) were unable to confirm the claims of Cambier regarding the fixation of ammonia by activated sludge, although they did find a marked "at once" action of activated sludge upon sewage. The average improvement, measured in terms of permanganate oxygen consumed, was about 29 percent when the period of contact was reduced to the minimum consistent with good mixing. "We think that the ability to produce a stable supernatant or filtrate as an immediate result of mixing activated sludge with sedimented sewage might constitute a test of activity of activated sludge."

Herb (1928) found that the oxidizability of the supernatant liquor was reduced by about 16 percent when a sewage was aerated for 5 minutes in the presence of 25 percent of activated sludge by volume. A steady state corresponding to 50 percent reduction was reached in about 30 minutes.

Theriault and McNamee (1930) reported a reduction of about 65 percent in the oxygen demand of a sewage following 10 minutes of agitation with a biological slime under laboratory conditions. This corresponds to the 90-percent reduction in 20 minutes observed under plant conditions.

Parsons (1929) could not obtain more than a slight "instantaneous effect" in his sewage clarification experiments using activated sludge as an absorbent. He suggests that the results of Butler and Coste may be in error because of an intervening filtration. Parsons, nevertheless, did obtain a marked improvement in his equilibrium experiments using a 30-minute period of agitation.

Grant, Hurwitz, and Mohlman (1930, p. 240) present a very consistent set of absorption curves, indicating a reduction of about 80 percent in oxygen requirements in 40 minutes, with little change when the aeration of the sludge-sewage mixtures was extended to 160 minutes.

Seiser (1928) concluded that the absorptive capacity of fully aerated activated sludge is largely exhausted in less than 1 hour.

The discrepancies in the graded series of observations just reviewed are probably more apparent than real. Negative results for the removal of ammonia, even though organic matters were readily removed, are satisfactorily explained by Butler and Coste (1927) on the basis that the sludge used in their experiments was drawn from a plant in which the second or nitrification stage of biological oxidation had not yet been fully established. As shown by complete deoxygenation curves, ammonia was not removed in the laboratory

experiments of Theriault and McNamee (1930) using sludge drawn from the upper sections of their artificial stream. Ammonia was readily removed, however, at "downstream" sections of the same installation.

Values reported by different observers for the percentage removal of organic matter from a given sewage cannot, of course, be compared without reference to the strength of the sewage, the absorptive capacity of the sludge, and other factors. The "at once" effect noted by Barraclough, and also by Butler and Coste, does not exclude later manifestations of the same phenomenon continued at a diminishing rate as the exhaustion of clarifying power is approached.

Negative results are seldom reported in the literature but they nevertheless have generally been obtained by the writer in preliminary experiments with sludges which are now believed to have been already fully saturated with sewage matters. It should obviously be desirable to work with fully activated or regenerated material in any attempted demonstration of the clarification process. With these explanations, it must be concluded that the evidence regarding the existence of a very rapid process of clarification is reasonably definite. If the phenomenon in question does occur with a given sludge, it should be completed in 30 or 40 minutes when pushed to the limit of the absorptive capacity of the sludge.

It is necessary to keep this definition clearly in mind so as to distinguish between clarification, as such, and the subsequent reactivation of the sludge or the regeneration of its clarifying power through the biological oxidation of absorbed materials. The statement that "2 or 3 hours are required for clarification by the activated sludge process" should be interpreted as including time for the period of regeneration. Clarification in the sense of turbidity removal, as by bacteria-eating plankton, is yet another phenomenon which is probably never completed in 30 or 40 minutes. On the other hand, the clarifying action of chemical precipitants may be regarded as closely related to the phenomenon under consideration. At all events the term "clarification" will be used only in the relative sense and not in the absolute sense with its connotation of complete removal of turbidity. A sewage may still remain quite turbid when clarified to the extent of 29 percent as in the experiments of Butler and Coste.

MECHANISM OF SEWAGE CLARIFICATION

Having shown the relation of clarification to the general theory of sewage treatment, and having presented the evidence regarding the validity of this useful concept, it will now be of interest to look more closely into some of the suggested explanations of the associated phenomena.

Mention has already been made of the explanation offered by Biltz and Krohnke for the efficacy of ferric salts in the precipitation of sewage colloids which, on the basis of migration experiments, had been shown to be negatively charged. It is to be noted that these experiments refer to the sewage colloids and not to the sludge. Surface attraction was suggested by Dunbar, and also by Jones and Travis, as a mechanism for the removal of the coarser or suspended matters by filter materials or by deposited sludge.

Theories of mutual coagulation of sewage colloids by the neutralization of electrical charges have frequently been advanced, in distinction to precipitation by outside agencies. With due regard to conventions regarding electrical charges, Cavel (1931), for example, reports that negative colloids, such as Congo red, are absorbed by activated sludge. Hence the sludge must be positively charged and, by further inference, the sewage colloids should be negatively charged. Dienert (1922) is less definite in his statement that the sewage colloids seem to be negatively charged while the sludge itself appears to be positively charged. Buswell (1928, p. 319) says: "One serious objection to the colloidal theory of coagulation is that the colloidal particles in sewage and the activated sludge particles are, so far as we are able to determine, both negatively charged. Since adsorption of colloids is most effective between oppositely charged particles, it should not be applied to the conditions of the activated sludge particles without reservation." Baly (1931) considers that, were it not for this observation of Buswell and Long (1923), the most attractive theory concerning the mutual coagulation of sewage and sludge colloids by the neutralization of electrical charges, "would seem to be one that could offer a completely satisfactory explanation of the activated sludge and activated filter bed processes of purification." To avoid this difficulty Baly (1931) has proposed a theory in which, as reported by Lumb (1933), it is assumed "that weakly charged colloidal particles will associate with strongly charged bacteria of the same sign, because the free energy of the whole system is thereby decreased." It should be incorrect to conclude that, by one theory or another, sewage colloids will always be mutually coagulated, regardless of sign.

In criticism of the colloidal theories of sewage precipitation it should be recalled that the suspended matters of sewage are by no means all of colloidal dimensions. Moreover, the colloidal theories at best cannot account for the removal of the considerable proportion of the nitrogenous and other oxygen-consuming impurities which are present in true solution. The presence of bacteria in the sludge particles should not be regarded as evidence of a transfer of the micro-organisms from the sewage to the sludge. Local growth on the favorable medium afforded by the sludge is to be considered.

The actual removal of bacteria from the sewage liquor may be largely accomplished by bacteria-eating plankton. It is not clear, moreover, that the previous history of the sample has always been considered in determining the character of electrical charges on activated sludges. The pH value of the liquid is often disregarded and the magnitude of the electrical charge appears to have been generally ignored, except by Baly.

From another angle it must be considered that the Baly theory, as applied to sewage colloids, does offer an explanation for the agglomeration of sewage colloids akin to the growth of drops by distillation from smaller to larger ones or, for that matter, to the growth of precipitates, as in water purification, from the stage of barely recognizable turbidity to the condition of visible "flocs."

In this case there can be no question as to the phenomenon itself and, furthermore, it must be considered that all of the particles composing the flocs originally possessed the same electrical sign. It appears reasonable, therefore, to assume a similar mechanism for the agglomeration of sewage colloids into sludge particles. Separate consideration should be given to the absorption of other organic matters by the sludge particles.

The biological theories of clarification are so intermingled with notions of oxidation that mechanisms for the rapid removal of organic matters from sewage are frequently obscured, although Johnson (1914, pp. 130–1), with reference to the action of the upper portion of a filter receiving crude sewage, is reasonably definite in his statement that "The filter material rapidly becomes coated with a slimy or gelatinous growth of *Zooglea ramigera*, which may be regarded as a large number of bacteria embedded in a gelatinous matrix." The gelatinous character of these zoogleal formations enables them to "absorb *soluble* polluting substances—as already described in Dunbar's absorption theory." The functions of a different class of filter organisms are emphasized by Buswell (1928, p. 331): "As indicated in the previous discussion, there are two groups of reactions which take place in trickling filters, (*a*) bioprecipitation and, (*b*) oxidation, or nitrification, as the latter is usually called. Bioprecipitation is accomplished by the larger organisms, the chlamydobacteriaceae or 'giant bacteria', the fungi ('especially molds'), the larger protozoa, and certain higher animals. These organisms are found to compose the bulk of the growths on the filter stones. A certain amount of oxidation is accomplished incidentally by these organisms, since they give off carbon dioxide."

With particular reference to the activated sludge process, the "Bioprecipitation Theory" is described as follows: "In view of previous work of other authors cited and the data of the present paper, we wish to propose the following statement of the theory of the activated

sludge process. Activated sludge flocs are composed of a *synthetic* gelatinous matrix similar to that of Nostoc or Merismopedia, in which filamentous and unicellular bacteria are embedded and on which various protozoa and some metazoa crawl and feed. The purification is accomplished by ingestion and assimilation by organisms of the organic matter in the sewage and its resynthesis into the living material of the flocs. This process changes organic matter from colloidal and dissolved states of dispersion to a state in which it will settle out" (Buswell and Long, 1923). Buswell (1931) describes activated sludge as follows: "The animal inclusions of the sludge made up a very small part of the entire mass. The base of the sludge was composed of zoogleal masses intermixed largely with filamentous bacteria and occasional *Zooglea ramigera.*" The surface area afforded by the zoogleal masses is estimated to be at least 500 square feet per cubic foot of aeration chamber. The importance of the zoogleal masses of bacteria has also been pointed out by Taylor (1930), and more recently the cultural and other characteristics of these gelatinous formations have been studied by Butterfield (1935) in this laboratory.

Buswell (1928, pp. 318–319) states that "there is practically no absorbed precipitated or coagulated amorphous matter in these sludge particles, but that they are composed entirely of active, growing microscopic organisms * * *. From what we know of the metabolism of micro-organisms it is probable that the unicellular forms absorb through their membrane such soluble forms of organic matter as are able to pass through this membrane, and that they also secrete enzymes which are capable of peptizing or liquefying colloidal particles too large to be directly absorbed. Protozoa, on the other hand, can easily be seen to approach and ingest visible particles of organic matter." The claim that activated sludge contains "practically no absorbed, precipitated, or coagulated amorphous matter" evidently refers to the fully activated material and not to sludge recently exposed to sewage matter. Likewise, the assumption that the sludge particles "are composed entirely of active, growing microscopic organisms" implies that the zoogleal masses, as a whole, are endowed with animate energy. As these gelatinous masses have been studied only bacteriologically, such a concept of zoogleal activity, although generally accepted, has probably accrued by default.

Parsons (1929) proposed an enzymatic theory coupled with "direct cell action" in explanation of the rapid removal of organic matters in sewage clarification. The protozoa are not mentioned, and as it appears unlikely that colloidal matter is taken up directly by bacteria, enzymes are introduced to account for the breaking down of larger particles. Substances in true solution are then transferred to the living organisms by osmosis. Parson's hypothetical enzyme, like

Buswell's is a lytic agent and not the clotting enzyme of the earlier chemists.

"Direct cell action" is assumed in Parson's theory of clarification as well as in the older theories of biological action. It is difficult, however, to visualize "direct cell action" by bacteria, because of the intervening gelatinous matrix in which most of the bacteria are embedded. On the other hand, "direct cell action" by the protozoa, etc., is an undeniable fact. Thus it is certain that to some extent the coarser particles of raw sewage are attacked and disintegrated by various species of plankton. Furthermore, the comminuted material may be visibly ingested by these relatively large organisms. By "coarser particles" in this connection are meant particles barely visible to the unaided eye. Suspended matters of greater dimension are, of course, infrequent even in crude sewage and they should be absent from the screened or settled sewage actually applied to filters or mixed with activated sludge.

Opinions as to the importance of protozoa in the clarification of sewage have ranged from the emphatic views of their usefulness expressed by Cramer (1931) to the suggestion by Fairbrother and Renshaw (1922) that the protozoa in activated sludge might be eliminated through the use of certain dyes because of their interference with the process. Butterfield, Purdy, and Theriault (1931) have held that the primary function of the protozoa is to prevent the bacterial population from reaching a stalemate, thereby stimulating bacterial growth with accompanying oxidation. The validity of the more restricted theories of biological action is nevertheless conditioned by the fact that the clarification of sewage, as defined in this paper, cannot very well be credited to the activities of either the bacteria or the protozoa. The action of the protozoa in the disintegration of coarser food particles and in the removal of bacteria from the sludge liquor must be regarded as a continuing process which is certainly not circumscribed by any 30-minute limitation. In the practical absence of quantitative data, the most favorable view of the matter would be to credit the protozoa with the removal of the excess population of bacteria from the body of the liquid. In this sense the protozoa should be credited with a secondary clarifying effect. The disintegration of coarser particles is probably accomplished by the protozoa only after these suspended matters have been agglomerated as a result of the primary clarification of the sewage.

As to the action of enzymes in the disintegration of coarser particles, it is tempting to replace Parson's hypothetical lytic agent by the protozoa, although bacterial enzymes are specifically mentioned by him in his important paper. Endoenzymes may account for the intracellular liquefaction or lysis of ingested food particles. There

is no evidence, however, of the existence of exocellular enzymes capable of liquefying suspended matter in 30 minutes or thereabouts. From the nature of the sludge floc any enzymes liberated by the bacteria, unless greatly diluted, should be stored in the gelatinous matrix. Lytic action should only be expected after the suspended matters have been collected on the floc. It should also be considered that the definite isolation of enzymes from the sewage or sludge has never been accomplished.

Theriault and McNamee (1930), in experiments already referred to, investigated the possibility that the extraordinary rate of apparent purification or clarification which obtained in their artificial stream was in reality an oxidation induced by the presence of a relatively high concentration of enzymes in the biological slime (sludge mat) deposited on the stream bed. The hypothetical enzyme visualized in these experiments was therefore an oxidase and not a clotting nor a lytic agent. Clarification by bacteria, or by combinations of bacteria and plankton, had been ruled out by known facts concerning their rates of oxidation. Reasons existed, however, for suspecting the existence of a more rapid oxidation process, loosely described in terms of the then ill-defined "immediate" oxygen demand of sewage and of sludges and tentatively ascribed to enzymatic action. Using the apparatus described by Theriault and Butterfield (1929), complete deoxygenation curves were accordingly obtained from which the respective rates of satisfaction of the "immediate" oxygen demand and of the oxygen demand related to biological activity could be deduced.

TABLE 1.—*Oxygen demand of sludge*

Period of incubation	Hours									
	1	2	4	6	11	23.5	29.5	47	74	95
	OXYGEN DEMAND—P. P. M.									
Rapid process (Y_2)	26	49	86	113	154	185	188	190	190	190
Slow process (Y_1)	8	16	32	48	87	177	217	325	465	556
Sum [1] (Y)	34	65	118	161	241	362	405	515	655	746
Observed	35	59	120	150	246	356	399	513	663	740

[1] Calculated by the formula,

$$Y = Y_2 + Y_1 = 190.3\,(1 - 10^{-0.093771}) + 1088.2\,(1 - 10^{-0.00345}).$$

As shown in table 1, the disappearance of atmospheric oxygen could very accurately be described in terms of two concurrent reactions, the slowest one corresponding to the normal rate of bacterial oxidation and reaching an asymptote in 20 days, and a much faster reaction which, however, was only completed in 20 hours or thereabouts. The results were therefore disappointing in respect to the existence of a major oxidation process which proceeded to 90 percent

completion in 20 minutes. The experimental conditions in these experiments were such that the conclusions as to the rates of oxidations could be checked by striking a very satisfactory balance between the observed decrease in the organic matter content of the sewage and the increased oxygen demand of the sludge mat. It is significant that the rate of bacterial oxidation deduced from these experiments accords satisfactorily with that observed in repeated experiments with river waters. The rate of satisfaction of the so-called "immediate" demand does not exclude certain types of enzymatic action. The assumption regarding the presence of exo-cellular enzymes in the sludge mat therefore remains a plausible but undemonstrated possibility. The existence of this relatively rapid process of oxidation is undeniable, and the same effect has since been repeatedly observed with sludge drawn from an activated sludge plant.

Absorption effects were invoked by Theriault and McNamee (1930) in explanation of the rapid clarification of sewage by activated sludges.

SUMMARY

It is apparent from the foregoing review that definiteness of statement in regard to the various phenomena of sewage treatment implies a recognition of the all-important element of time. With the information at hand regarding underlying rates, it has become possible clearly to separate the basic phenomena of sewage treatment into processes which approach completion, respectively, in days, hours, or minutes. On this basis, it is evident that the participation of the biological elements as a major factor in the primary clarification of sewage is definitely ruled out, unless the difficult hypothesis is introduced of direct absorption by bacteria, etc., without accompanying oxidation. If the clarification process is referred to enzymatic action or to the activities of protozoa, it is still necessary to provide a mechanism for the rapid transfer of organic matters to the enzyme substrate or to the protozoan nidus, in either case, the sludge floc.

From various angles, therefore, attention should be focused on the floc itself as the primary absorbent, apart from embedded bacteria, secreted enzymes, or attending protozoa. Such a view of sewage clarification, while plausible enough, raises a difficult question in regard to the nature of the gelatinous matrix which has hitherto been generally regarded as an indissoluble component of the bacteria. New light on this seemingly intractable problem is afforded by the recent announcement (PUBLIC HEALTH REPORTS, Feb. 1, 1935) that the adsorbent principle in activated sludge had been definitely identified as a base-exchanging substance, chemically the same as the zeolites of water purification. The data substantiating this proximate analysis of the gelatinous matrix in activated sludge will be

presented in the next paper of this series. A discussion of numerous and more or less obvious implications as regards the theory or practice of sewage treatment will be given in succeeding papers.

REFERENCES

Ardern, E. (1921): Sewage purification with reference to colloid chemistry. British Assoc. Adv. Science, 2nd Report on Colloid Chemistry, pp. 81–95.

Baly, E. C. C. (1931): The mechanism of the activated sludge process of sewage disposal. J. Soc. Chem. Ind., **50**, 22–6T.

Biltz, W., and Krohnke, O. (1904): Ueber organische Colloide aus städtischen Abwässern und deren Zustandsaffinität. Ber., **37**, 1745–54.

Buswell, A. M., and Long, H. L. (1923): Microbiology and theory of activated sludge. J. Am. Water Works Assoc., **10**, 309–21.

Buswell, A. M. (1928): The Chemistry of Water and Sewage Treatment. American Chem. Soc. Monograph Ser. No. 38. New York.

Buswell, A. M. (1931): The biology of activated sludge. An historical review. Sew. Works J., **3**, 362–8.

Butler, W., and Coste, J. H. (1927): Modern methods of sewage disposal. J. Soc. Chem. Ind., **46**, 49–59T.

Cambier, R. (1920): Sur l'épuration des éaux d'égout par les boues activées. Compt. rend. Acad. d. sc., Par. **170**, 681–4.

Cavel, L. (1931): Sur l'adsorption des matiéres colloïdales par les "boues activées. Rev. d'Hyg. et Med. Prev., **53**, 179–81.

Cramer, R. (1931): The role of protozoa in activated sludge. Ind. Eng. Chem., **23**, 309–13.

Dienert, F. (1922): Épuration des eaux d'égout par les boues activées. Rev. d'Hyg., **44**, 113–66.

Dunbar, W. P. (1907): Principles of Sewage Treatment (translated by H. T. Calvert). C. Griffin & Co., Ltd., London.

Fairbrother, T. H., and Renshaw, A. (1922): The relation between chemical constitution and antiseptic action in the coal-tar dyestuffs. J. Soc. Chem. Ind., **41**, 134–44T.

Frankland, E. (1868): First report rivers pollution commission (England).

Grant, S., Hurwitz, E., and Mohlman, F. W. (1930). The oxygen requirements of the activated sludge process. Sew. Works J., **2**, 228–44.

Herb, O. (1928): Absorptionsversuche mit belebtem Schlamm. Arch. Hyg., **100**, 112–20.

Johnson, J. W. H. (1914): A contribution to the biology of sewage disposal. J. Econ. Biol., **9**, 105–25, 127–63.

Jones, A. S., and Travis, W. O. (1906): On the elimination of suspended solids and colloidal matters from sewage. Proc. Inst. Civil Eng. (London), **164**, 68–94.

Lumb, L. (1933): Some notes on the mechanism of the activated sludge process. J. Inst. Sewage Purification, Part I, July, pp. 21–32.

Parsons, A. S. (1929): Notes on the clarification stage of the activated sludge process. Surveyor, **72**, 221–6; *see also* Water Works and Sewerage, **76**, 397–9 (1929), and Can. Eng., **58**, 125 (1930).

Seiser, A. (1928): (Research on the mechanism of the activated sludge process). Gesundh. Ing., **51**, 253–9, 273–6; reviewed in Sew. Works J., **1**, 265–7 (1929).

Stoddart, F. W. (1909): Nitrification and the absorption theory. Proc. 7th Int. Congr. App. Chem., Section VIIIa, pp. 183–210.

Taylor, H. (1930): Some biological notes on sewage disposal processes. Surveyor, **73**, 32–3.

Theriault, E. J., and Butterfield, C. T. (1929): Experimental studies of natural purification in polluted waters. I. Apparatus and technique for the study of biochemical and other oxidations in liquids. Pub. Health Rep., **44**, 2253–67. (Reprint No. 1317.)

Theriault, E. J., and McNamee, P. D. (1930): Sludge aeration experiments. I. Rate of disappearance of oxygen in sludge. Ind. Eng. Chem., **22**, 1330–6; Pub. Health Rep., **46**, 1301–19. (Reprint No. 1480.)

Wilson, H. (1930): Colloids in relation to sewage purification. Proc. Assoc. Managers Sewage Disposal Works, pp. 171–87.

Young, M., and Melling, S. E. (1918): Activated sludge in the treatment of sewage. J. Roy. San. Inst., **39**, 10–16; see discussion, p. 19

DEATHS DURING WEEK ENDED OCT. 19, 1935

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Oct. 19, 1935	Corresponding week, 1934
Data from 86 large cities of the United States:		
Total deaths	7,865	7,836
Deaths per 1,000 population, annual basis	11.0	10.9
Deaths under 1 year of age	493	597
Deaths under 1 year of age per 1,000 estimated live births	45	56
Deaths per 1,000 population, annual basis, first 42 weeks of year	11.4	11.3
Data from industrial insurance companies:		
Policies in force	67,783,476	67,015,611
Number of death claims	11,498	12,803
Death claims per 1,000 policies in force, annual rate	8.8	10.0
Death claims per 1,000 policies, first 42 weeks of year, annual rate	9.7	9.9

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers

Reports for Weeks Ended Oct. 26, 1935, and Oct. 27, 1934

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Oct. 26, 1935, and Oct. 27, 1934

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934
New England States:								
Maine	----	2	----	1	45	4	0	0
New Hampshire	----	----	----	----	----	1	0	0
Vermont	3	----	----	----	32	2	0	0
Massachusetts	11	17	----	----	53	27	5	1
Rhode Island	----	3	----	----	4	----	0	0
Connecticut	6	3	----	1	58	51	0	0
Middle Atlantic States:								
New York	34	46	[1] 7	[1] 10	234	122	6	3
New Jersey	20	10	3	12	23	19	0	0
Pennsylvania	49	70	----	----	53	256	4	2
East North Central States:								
Ohio	95	111	9	45	58	104	3	0
Indiana	99	100	27	28	6	65	3	1
Illinois	84	118	16	8	20	132	5	3
Michigan	8	16	3	2	22	37	1	2
Wisconsin	5	8	26	6	55	117	1	2
West North Central States:								
Minnesota	6	10	----	1	14	60	2	0
Iowa	28	18	3	----	2	11	2	2
Missouri	93	87	42	17	26	72	3	1
North Dakota	7	4	----	----	3	33	0	0
South Dakota	4	----	----	----	3	4	0	0
Nebraska	16	16	----	----	40	1	0	0
Kansas	12	3	3	2	3	50	0	0
South Atlantic States:								
Delaware	----	4	----	1	56	----	0	0
Maryland [2]	10	9	6	16	7	9	2	2
District of Columbia	18	8	1	----	----	2	4	0
Virginia	82	164	----	----	15	118	4	1
West Virginia	66	92	16	18	----	46	2	0
North Carolina	124	138	8	8	----	30	2	1
South Carolina	13	----	185	----	6	2	3	0
Georgia [3]	57	71	----	----	----	----	0	0
Florida [3]	24	14	----	----	1	2	0	0

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Oct. 26, 1935, and Oct. 27, 1934—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934
East South Central States:								
Kentucky	74	78	9	11	65	4	0	2
Tennessee [3]	72	80	22	18	2	43	4	0
Alabama [3]	45	62	36	17	9	26	1	0
Mississippi [3]	36	27	----	----	----	----	0	0
West South Central States:								
Arkansas	22	23	12	4	2	----	0	2
Louisiana	28	38	12	3	13	11	0	2
Oklahoma [4]	25	19	16	28	2	1	2	3
Texas [3]	170	75	153	129	7	39	3	3
Mountain States:								
Montana	4	4	5	4	10	47	0	0
Idaho	1	10	2	3	----	----	0	1
Wyoming	1	----	----	----	18	3	0	0
Colorado	18	11	----	----	6	48	0	2
New Mexico	7	3	2	4	8	19	0	0
Arizona	8	1	29	5	2	20	0	0
Utah [3]	1	----	----	----	1	5	0	0
Pacific States:								
Washington	3	2	----	----	88	106	1	0
Oregon	1	----	21	23	108	7	1	0
California	65	42	24	19	137	156	2	2
Total	1,555	1,617	698	444	1,317	1,912	66	38
First 43 weeks of year	27,581	29,823	108,928	53,887	702,700	678,037	4,793	1,937

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934
New England States:								
Maine	6	2	22	15	0	0	2	9
New Hampshire	0	0	9	9	0	0	0	0
Vermont	4	0	5	9	0	0	1	1
Massachusetts	35	2	120	122	0	0	2	2
Rhode Island	7	0	12	8	0	0	1	1
Connecticut	9	0	44	25	0	0	1	2
Middle Atlantic States:								
New York	45	7	271	251	0	0	10	26
New Jersey	22	0	68	91	0	0	1	5
Pennsylvania	1	4	291	338	0	0	14	17
East North Central States:								
Ohio	0	6	290	388	2	0	25	36
Indiana	4	3	125	121	3	2	7	12
Illinois	12	15	392	343	5	6	20	32
Michigan	14	5	139	196	0	1	9	16
Wisconsin	1	17	275	365	4	7	4	10
West North Central States:								
Minnesota	0	1	151	57	0	16	3	1
Iowa	1	1	82	50	4	3	10	10
Missouri	1	0	143	70	0	0	16	17
North Dakota	1	0	28	30	0	0	8	4
South Dakota	2	1	42	9	12	0	1	0
Nebraska	0	0	35	21	5	3	0	0
Kansas	1	4	88	47	2	3	5	4
South Atlantic States:								
Delaware	0	0	4	11	0	0	4	4
Maryland [3]	1	0	81	106	0	6	16	9
District of Columbia	3	0	13	18	0	0	2	3
Virginia	4	4	68	127	0	2	29	22
West Virginia	0	2	173	155	0	0	10	25
North Carolina	3	0	135	151	0	1	8	6
South Carolina	1	0	7	15	0	0	5	12
Georgia [3]	0	1	33	33	0	0	13	7
Florida [3]	1	0	7	4	0	0	0	3

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Oct. 26, 1935, and Oct. 27, 1934—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934	Week ended Oct. 26, 1935	Week ended Oct. 27, 1934
East South Central States:								
Kentucky	7	3	77	76	0	0	12	19
Tennessee [2]	1	1	87	93	0	0	13	26
Alabama [2]	1	3	14	33	0	0	11	14
Mississippi [2]	0	0	16	27	1	1	6	6
West South Central States:								
Arkansas	0	0	18	8	0	1	4	13
Louisiana	3	1	14	16	1	1	33	31
Oklahoma [4]	0	0	21	13	0	0	18	22
Texas [3]	3	7	78	37	0	1	32	35
Mountain States:								
Montana	0	12	52	38	10	0	1	3
Idaho	0	3	20	2	0	1	1	2
Wyoming	0	0	14	1	0	1	0	0
Colorado	0	0	107	99	0	0	3	4
New Mexico	0	0	6	15	0	0	33	26
Arizona	0	0	11	14	0	0	2	2
Utah [2]	1	0	52	17	0	0	0	2
Pacific States:								
Washington	5	25	41	46	31	17	6	7
Oregon	2	2	30	75	0	0	4	2
California	21	31	190	196	0	0	14	13
Total	223	163	4,001	3,991	80	67	420	523
First 43 weeks of year	9,839	6,650	202,863	170,269	5,686	4,140	15,351	18,054

[1] New York City only.
[2] Week ended earlier than Saturday.
[3] Typhus fever, week ended Oct. 26, 1935, 29 cases, as follows: Georgia, 13; Florida, 1; Tennessee, 1; Alabama, 4; Texas, 10.
[4] Exclusive of Oklahoma City and Tulsa.

Summary of Monthly Reports from States

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
August 1935										
Colorado	3	31	------	------	23	------	3	65	1	29
September 1935										
California	13	129	61	54	325	7	101	416	4	79
Florida	------	34	4	56	11	2	1	23	0	13
Kansas	2	45	3	4	18	------	7	183	30	35
Louisiana	1	91	51	689	24	8	6	28	0	108
Mississippi	3	123	995	10,012	26	324	2	77	2	33
Montana	1	5	48	------	14	------	2	123	------	12
New Hampshire	------	------	------	------	------	------	------	14	0	4
New York	40	98	------	8	290	------	1,054	568	------	147
Oklahoma [1]	6	79	102	539	4	10	1	50	1	83
Oregon	------	------	30	13	210	------	4	97	1	5
Rhode Island	1	4	------	------	15	------	137	33	0	2
Tennessee	19	164	111	470	21	31	14	186	1	158
Texas	2	265	89	4,020	18	28	6	92	2	208
Virginia	9	114	245	79	25	8	64	97	76	6
Washington	3	8	15	------	83	1	------	109	28	16
Wisconsin	4	14	98	------	173	------	16	427	6	19

[1] Exclusive of Oklahoma City and Tulsa.

August 1935

	Cases
Colorado:	
Chicken pox	19
Epidemic encephalitis	1
Impetigo contagiosa	2
Mumps	57
Paratyphoid fever	2
Rocky Mountain spotted fever	1
Whooping cough	51

September 1935

	Cases
Actinomycosis:	
California	2
Anthrax:	
Oklahoma [1]	1
Botulism:	
California	2
Chicken pox:	
California	280
Florida	4
Kansas	56
Mississippi	148
Montana	50
New York	211
Oklahoma [1]	8
Oregon	27
Rhode Island	1
Tennessee	15
Texas	4
Virginia	37
Washington	114
Wisconsin	224
Dengue:	
Florida	1
Mississippi	11
Texas	6
Dysentery:	
California (amœbic)	11
California (bacillary)	27
Florida (bacillary)	4
Kansas (bacillary)	1
Louisiana (amœbic)	2
Mississippi (amœbic)	88
Mississippi (bacillary)	368
Montana (bacillary)	2
New York (amœbic)	4
New York (bacillary)	91
Oregon (amœbic)	3
Tennessee (bacillary)	13
Texas (amœbic)	3
Texas (bacillary)	3
Virginia (diarrhea included)	234
Epidemic encephalitis:	
California	9
Kansas	9
New York	14
Oklahoma [1]	1
Oregon	1
Texas	2
Virginia	3
Wisconsin	2
Food poisoning:	
California	19
German measles:	
California	166
Kansas	6
Montana	4
New York	72
Washington	33
Wisconsin	50

September 1935—Continued

	Cases
Hookworm disease:	
California	1
Louisiana	5
Mississippi	301
Impetigo contagiosa:	
Kansas	5
Oklahoma [1]	4
Oregon	35
Tennessee	15
Washington	6
Mumps:	
California	483
Florida	24
Kansas	128
Mississippi	153
Montana	62
Oklahoma [1]	6
Oregon	37
Rhode Island	59
Tennessee	15
Texas	111
Virginia	33
Washington	125
Wisconsin	676
Ophthalmia neonatorum:	
New York	3
Oklahoma [1]	2
Tennessee	2
Virginia	2
Paratyphoid fever:	
California	5
Florida	3
Kansas	3
New York	10
Oregon	5
Tennessee	7
Texas	13
Virginia	6
Puerperal septicemia:	
Mississippi	27
Tennessee	5
Washington	1
Rabies in animals:	
California	49
Kansas	3
Louisiana	18
Mississippi	2
New York [2]	2
Oregon	1
Texas	20
Washington	1
Rabies in man:	
Washington	1
Relapsing fever:	
California	2
Rocky Mountain spotted fever:	
Tennessee	1
Virginia	2
Scabies:	
Oregon	47
Tennessee	10
Septic sore throat:	
California	6
Kansas	6
Louisiana	3
Montana	24
New York	23
Oklahoma [1]	45
Rhode Island	3

September 1935—Continued

	Cases
Septic sore throat—Con.	
Tennessee	5
Virginia	1
Washington	4
Wisconsin	4
Tetanus:	
California	5
Kansas	4
Louisiana	4
New York	1
Rhode Island	1
Virginia	2
Trachoma:	
California	10
Kansas	4
Mississippi	9
Montana	46
Oklahoma [1]	3
Tennessee	26
Virginia	1
Wisconsin	2
Trichinosis:	
California	6
New York	11
Oregon	1
Tularaemia:	
California	32
Louisiana	1
Tennessee	1
Texas	1
Virginia	3
Typhus fever:	
Florida	4
New York	3
Texas	24
Virginia	1
Undulant fever:	
California	17
Florida	1
Kansas	4
Louisiana	2
New York	23
Oklahoma [1]	1
Rhode Island	1
Tennessee	3
Texas	2
Washington	3
Wisconsin	8
Vincent's infection:	
Kansas	3
New York [2]	69
Oklahoma [1]	1
Oregon	5
Tennessee	6
Whooping cough:	
California	425
Florida	16
Kansas	150
Louisiana	12
Mississippi	232
Montana	131
New York	1,270
Oklahoma [1]	35
Oregon	30
Rhode Island	59
Tennessee	142
Texas	118
Virginia	52
Washington	49
Wisconsin	667

[1] Exclusive of Oklahoma City and Tulsa.
[2] Exclusive of New York City.

WEEKLY REPORTS FROM CITIES

City reports for week ended Oct. 19, 1935

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Maine:											
Portland	0	------	1	0	1	0	0	0	0	15	30
New Hampshire:											
Concord	0	------	0	0	1	1	0	1	0	0	12
Manchester	0	------	1	0	0	0	0	2	0	0	10
Nashua	0	------	--------	0	--------	2	0	--------	0	0	--------
Vermont:											
Barre	0	------	0	0	0	0	0	0	0	0	3
Burlington	0	------	0	0	0	0	0	0	0	0	7
Rutland	0	------	0	0	0	0	0	0	0	2	10
Massachusetts:											
Boston	2	------	0	4	12	27	0	7	3	10	179
Fall River	0	------	0	1	4	4	0	1	0	6	35
Springfield	0	------	0	1	0	4	0	3	0	9	44
Worcester	1	------	0	1	5	15	0	1	0	1	59
Rhode Island:											
Pawtucket	0	------	0	0	0	1	0	0	0	0	13
Providence	0	------	0	0	4	1	0	1	0	[1] 13	64
Connecticut:											
Bridgeport	1	1	1	0	2	3	0	1	1	0	38
Hartford	0	------	0	0	2	2	0	1	0	7	36
New Haven	1	------	0	0	0	1	0	0	0	4	35
New York:											
Buffalo	0	------	0	7	20	35	0	3	0	21	150
New York	27	10	3	32	100	76	0	90	17	111	1,428
Rochester	1	------	0	1	2	4	0	0	0	3	59
Syracuse	0	------	0	0	3	4	0	0	0	1	45
New Jersey:											
Camden	0	------	0	0	2	3	0	1	1	1	39
Newark	0	3	0	1	12	15	0	6	1	32	111
Trenton	1	------	0	0	3	4	0	1	0	2	42
Pennsylvania:											
Philadelphia	3	------	0	7	18	57	0	19	5	53	412
Pittsburgh	3	------	0	5	20	54	0	10	1	42	171
Reading	2	------	0	1	2	2	0	0	0	0	23
Scranton	0	------	--------	0	--------	3	0	--------	0	0	--------
Ohio:											
Cincinnati	7	------	0	2	3	13	0	6	0	10	124
Cleveland	11	15	1	1	8	28	0	12	2	44	155
Columbus	9	------	0	0	2	20	0	5	2	2	81
Toledo	1	------	0	0	6	7	0	2	0	3	59
Indiana:											
Anderson	0	------	0	0	0	1	0	2	0	1	12
Fort Wayne	11	------	0	0	5	8	0	0	0	0	21
Indianapolis	3	------	0	3	14	17	0	3	2	9	91
Muncie	0	------	0	3	2	2	0	0	0	0	8
South Bend	0	------	0	0	5	0	0	0	0	0	17
Terre Haute	0	------	0	0	0	1	0	0	0	0	22
Illinois:											
Alton	6	------	0	0	0	2	0	0	0	0	9
Chicago	7	5	0	9	32	101	0	35	1	76	633
Elgin	0	------	0	0	0	7	0	0	0	0	11
Moline	1	------	0	0	1	0	0	0	0	0	5
Springfield	1	------	0	0	2	1	0	0	0	1	24
Michigan:											
Detroit	7	3	0	3	17	32	0	16	1	112	255
Flint	1	------	0	0	4	15	0	1	0	2	36
Grand Rapids	0	------	0	0	1	5	0	0	0	4	32
Wisconsin:											
Kenosha	0	------	0	0	3	14	0	0	0	5	8
Milwaukee	0	1	1	1	7	28	0	5	0	73	90
Racine	1	------	0	1	0	27	0	0	0	12	8
Superior	0	------	0	0	0	1	0	1	0	0	6
Minnesota:											
Duluth	0	------	0	1	1	1	0	0	0	1	16
Minneapolis	10	------	0	5	9	55	0	1	1	8	94
St. Paul	0	------	0	0	7	21	0	1	0	5	61

[1] Including delayed reports.

City reports for week ended Oct. 19, 1935—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Iowa:											
Cedar Rapids	0			0		2	0		0	2	
Davenport	0			0		1	0		0	0	
Des Moines	0			0		5	0			1	33
Sioux City	1			0		3	0		0	2	
Waterloo	9			0		7	0		0	0	
Missouri:											
Kansas City	1		0	0	7	8	0	4	0	3	84
St. Joseph	3		0	0	2	1	0	1	0	0	27
St. Louis	21	3	3	2	4	43	0	8	0	2	210
North Dakota:											
Fargo	3		0	0	0	3	0	0	0	2	8
Grand Forks	0			0		0	0		0	0	
Minot	0		0	0	0	1	0	0	0	1	3
South Dakota:											
Aberdeen	0			0		0	0		0	0	
Nebraska:											
Omaha	13		0	0	2	15	0	1	0	0	54
Kansas:											
Lawrence	0		0	0	0	1	0	0	0	0	4
Topeka	0		0	0	2	3	0	0	0	1	13
Wichita	2		0	1	3	2	0	0	3	1	21
Delaware:											
Wilmington	1		0	0	3	0	0	1	1	1	28
Maryland:											
Baltimore	5	5	0	0	17	22	0	12	2	14	208
Cumberland	2		0	0	1	2	0	0	1	0	19
Frederick	0		0	0	0	1	0	0	0	0	1
District of Col.:											
Washington	6	1	1	0	7	14	0	9	2	2	190
Virginia:											
Lynchburg	1		0	0	0	1	0	0	0	0	9
Norfolk	0		0	0	7	1	0	0	0	0	35
Richmond	1		0	0	3	1	0	5	1	0	52
Roanoke	4		0	0	0	2	0	0	0	0	22
West Virginia:											
Charleston	3		0	0	0	8	0	0	0	0	9
Huntington	5			0		20	0		0	0	
Wheeling	0		0	1	3	7	0	0	1	0	22
North Carolina:											
Gastonia	2	2	0	0	1	0	0	0	0	0	5
Raleigh	1		0	0	1	0	0	1	0	0	17
Wilmington	1		0	0	0	0	0	1	0	0	10
Winston-Salem	1		0	0	1	4	0	0	1	0	13
South Carolina:											
Charleston	1	14	0	0	1	2	0	2	0	0	23
Columbia	0		0	0	0	0	0	0	0	0	4
Florence	0		0	0	1	0	0	0	0	0	8
Greenville	1		0	0	0	1	0	0	0	0	3
Georgia:											
Atlanta	5	4	1	1	5	9	0	6	0	4	78
Brunswick	0		0	0	0	0	0	0	0	0	2
Savannah	3		0	1	2	4	0	1	0	1	30
Florida:											
Miami	4	2	0	1	2	0	0	2	0	1	26
Tampa	0		0	0	0	2	0	0	0	0	19
Kentucky:											
Ashland	3	0		0		1	0		0	0	
Covington	3	0		0	2	7	0	0	0	1	13
Louisville	5	6	0	0	2	17	0	2	2	1	55
Tennessee:											
Knoxville	6	0		0	3	4	0	0	3	0	22
Memphis	1		2	0	5	8	0	6	0	2	83
Nashville	4		0	0	3	3	0	2	0	1	50
Alabama:											
Birmingham	2	4	0	0	4	3	0	1	0	0	60
Mobile	5		0	0	5	0	0	1	1	0	26
Montgomery	1			1		1	0		0	0	
Arkansas:											
Fort Smith	2			0		3	0		0	0	
Little Rock	0		0	1	5	2	0	1	0	0	6
Louisiana:											
Lake Charles	0		0	0		0	0		0	0	9
New Orleans	10	1	0	0	13	6	0	11	0	20	157
Shreveport	2		0	0	2	1	0	2	1	0	35

City reports for week ended Oct. 19, 1935—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Texas:											
Dallas	10	------	0	0	3	12	0	3	0	6	56
Fort Worth	10	------	0	------	1	9	0	1	1	------	38
Galveston	0	------	0	0	0	0	0	2	2	0	15
Houston	13	------	0	0	2	1	0	6	1	0	76
San Antonio	2	------	2	0	5	0	0	7	0	0	56
Montana:											
Billings	0	------	0	0	0	1	0	0	0	2	2
Great Falls	0	------	0	0	1	2	0	0	0	1	9
Helena	0	------	0	0	0	0	0	0	0	0	5
Missoula	0	------	0	0	1	24	0	0	0	0	5
Idaho:											
Boise	0	------	0	0	2	3	0	0	0	0	7
Colorado:											
Colorado Springs	0	------	1	0	1	6	0	2	0	1	14
Denver	2	------	1	2	4	16	0	4	0	0	75
Pueblo	0	------	0	2	0	13	0	0	0	1	13
New Mexico:											
Albuquerque	1	------	0	0	0	3	0	5	2	0	19
Utah:											
Salt Lake City	0	------	1	0	0	46	0	1	0	7	30
Nevada:											
Reno	0	------	0	0	0	1	0	0	0	0	2
Washington:											
Seattle	0	------	0	1	5	15	0	3	0	0	94
Spokane	0	------	0	2	5	5	3	0	0	0	24
Tacoma	------	------	------	4	------	2	------	------	------	------	------
Oregon:											
Portland	0	------	0	3	2	24	0	1	0	0	70
Salem	0	------	------	0	------	3	0	------	0	0	------
California:											
Los Angeles	16	21	0	5	8	22	1	19	6	8	264
Sacramento	5	------	0	1	1	9	0	1	------	9	22
San Francisco	0	2	0	30	7	5	0	6	1	22	179

State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths	
Maine:			
Portland	0	0	1
Massachusetts:			
Boston	1	1	13
Springfield	0	0	1
Worcester	0	0	4
Rhode Island:			
Providence	1	0	5
Connecticut:			
Hartford	0	0	4
New York:			
New York	10	3	39
New Jersey:			
Newark	0	0	2
Pennsylvania:			
Philadelphia	1	0	5
Pittsburgh	1	0	0
Ohio:			
Cincinnati	3	1	0
Columbus	0	1	0
Illinois:			
Chicago	2	1	1
Michigan:			
Detroit	1	1	1
Wisconsin:			
Kenosha	0	0	1
Milwaukee	0	0	1
Minnesota:			
Minneapolis	0	1	0
Missouri:			
St. Louis	3	1	1
South Dakota:			
Aberdeen	0	0	2
Maryland:			
Baltimore	2	1	2
District of Columbia:			
Washington	2	3	1
Virginia:			
Norfolk	[1] 2	0	0
Richmond	0	0	3
North Carolina:			
Raleigh	0	1	0
Kentucky:			
Louisville	0	0	6
Tennessee:			
Memphis	1	0	0
Nashville	0	3	1
Alabama:			
Birmingham	1	0	1
Louisiana:			
New Orleans	0	0	2
Shreveport	0	[1] 1	0
Texas:			
Dallas	1	1	0
Utah:			
Salt Lake City	0	0	1
Washington:			
Seattle	0	0	2
California:			
Los Angeles	0	0	6
Sacramento	1	0	0

[1] Imported.

Epidemic encephalitis.—Cases: Boston, 1; Newark, 1; Toledo, 1; Chicago, 1; Superior, 1.
Pellagra.—Cases: Washington, D. C., 1; Birmingham, 1; New Orleans, 1; Dallas, 1; Los Angeles, 3; San Francisco, 1.
Rabies in man.—Deaths: Raleigh, 1; Houston, 1.
Typhus fever.—Cases: Atlanta, 1; Montgomery, 1; New Orleans, 1; Houston, 3.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Two weeks ended October 5, 1935.—During the 2 weeks ended October 5, 1935, certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			1	4	2				1	8
Chicken pox				55	158	58	66	37	45	419
Diphtheria		3	8	25	14	16	5		2	73
Dysentery				13	3					16
Erysipelas				7	5	5		1	4	22
Influenza		15			12	3			38	68
Measles		4	69	117	248	2	30	6	186	662
Mumps		9			125	80	277	12	31	534
Paratyphoid fever	4	4			8				1	17
Pneumonia		3			12		1		6	22
Poliomyelitis					15	4	5	28	4	56
Scarlet fever	1	20	2	186	184	71	9	13	44	530
Smallpox							3		1	4
Trachoma						1	1		18	20
Tuberculosis	3	66	37	121	140	32	4	5	28	436
Typhoid fever	6	2	19	49	50	3	4	7	4	144
Undulant fever					3					3
Whooping cough		9	17	131	171	65	75	19	17	504

CZECHOSLOVAKIA

Communicable diseases—August 1935.—During the month of August 1935 certain communicable diseases were reported in Czechoslovakia, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	9		Paratyphoid fever	19	1
Cerebrospinal meningitis	7	5	Poliomyelitis	33	2
Chicken pox	22		Puerperal fever	35	12
Diphtheria	1,529	97	Scarlet fever	1,404	20
Dysentery	235	17	Trachoma	114	
Influenza	42	6	Typhoid fever	563	52
Lethargic encephalitis	2	1	Typhus fever	3	
Malaria	558				

PANAMA CANAL ZONE

Communicable diseases—July–September 1935.—During the months of July, August, and September 1935, certain communicable diseases, including imported cases, were reported in the Panama Canal Zone and terminal cities as follows:

Disease	July		August		September	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Chicken pox	1				4	
Diphtheria	9	1	17	2	14	1
Dysentery (amoebic)	26	1	35		32	2
Dysentery (bacillary)	3	1	1			
Leprosy	2			1		
Lethargic encephalitis			1			
Malaria	194	3	84	6	107	1
Measles	1				1	
Meningococcus meningitis	1		1	1	1	1
Mumps	2					
Paratyphoid fever			1			
Pneumonia		19		17		10
Poliomyelitis			1		1	
Tuberculosis		27		27		30
Typhoid fever	3		4	1	6	
Typhus fever	1					
Whooping cough	16		5		3	

VIRGIN ISLANDS

Notifiable diseases—July–September 1935.—During the months of July, August, and September 1935, cases of certain notifiable diseases were reported in the Virgin Islands as follows:

Disease	July	August	September	Disease	July	August	September
Chicken pox	1	1		Malaria	2	3	2
Filariasis	6	3	4	Poliomyelitis	1	1	
Fish poisoning	2			Syphilis	21	14	3
Gonorrhea	2	9	11	Tuberculosis	2	3	
Hookworm disease	2	2	2				

YUGOSLAVIA

Communicable diseases—September 1935.—During the month of September 1935 certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	121	6	Poliomyelitis	4	
Cerebrospinal meningitis	7	3	Scarlet fever	449	5
Diphtheria and croup	607	61	Sepsis	2	1
Dysentery	705	79	Tetanus	57	18
Erysipelas	231	5	Typhoid fever	766	69
Measles	63	1	Typhus fever	11	
Paratyphoid fever	47	1			

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for October 25, 1935, pages 1512–1526. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued November 29, 1935, and thereafter, at least for the time being, in the issue published on the last Friday of each month.

Plague

Hawaii Territory—Hawaii Island—Hamakua District—Paauhau Sector.—A rat which was found October 14, 1935, in Paauhau Sector, Hamakua District, Island of Hawaii, has been proved positive for plague. The rat was found in the course of plague eradicative work.

Yellow Fever

Gold Coast.—Yellow fever has been reported in Gold Coast as follows: On October 6, 1935, 3 cases at Bawku; September 21, 1935, 1 case and 1 death at Tamale.